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Psychological Monographs: General and Applied

Autonomic and Muscular Responses, and Their Relation to Simple Stimuli[1]

R. C. Davis, Alexander M. Buchwald, and R. W. Frankmann

Indiana University

I. SOMATIC RESPONSES AND PSYCHOLOGICAL THEORY

INVESTIGATORS might be forgiven for putting off any reflections on the significance of their enterprise until it is well advanced. But when the investigator faces many options, as he does in psychology, his choice of subject matter needs to be accounted for.

There is no difficulty in finding more than one line of thought in psychology which would provoke the investigation of autonomic responses and skeletal muscle tension; indeed "somatic responses," if we may appropriate the term for convenience, seem to play a rather important part in all sorts of psychological thought except that which holds that knowledge will best be advanced by ignorance of the transcutaneous. These somatic responses are often discussed and sometimes experimented on because they are thought to be the flesh-and-blood embodiments of certain immaterial realities which psychological analysis seems to discover: psychological concepts such as emotion, anxiety, stress, inner tension, subvocal speech, meaning, and fractional anticipatory goal responses, for example. Disappointments have been severe, and more of them are probably to come; but finding that somatic responses do not meet the demands of a theory is not enough reason for disowning them and deciding that the theory must apply to something else.

We propose to take a broader—and less directed—view. Rather than being hopeful of homologies between the somatic responses and psychological concepts built from other materials, we should be surprised by their discovery and rather astonished at the simplicity of the universe. Nevertheless, somatic responses abound. One has but to observe them on a set of recording instruments to believe that they are by far the most numerous responses of the organism. It is clear that any overt response, vocal utterance, or bodily movement, is surrounded by a wide penumbra of them, and it may not be too bold a guess to say that whenever there is any evidence of a stimulus affecting the individual, something in his periphery or viscera is set into motion. Not infrequently these may be seen when there is no other means at hand for detecting that the person has been stimulated. In point of time, the somatic responses may lead up to an overt response, accompany it, or follow it. In this sea of somatic response an occasional wave breaks into an external response. To understand these breaks we

[1] Work conducted under Contract Nonr-908(03), between Office of Naval Research and Indiana University. H. M. Madison assisted in the collection of data.

suppose that a study of the waves in general would be a great help. Or, to leave the figure of speech, we would say that confronted with problems about the last of a chain of events, we propose the study of events immediately before it, and of similar events which are not followed by the same conclusion.

These phrases may seem to mean that the immediate causes of the external action are in the somatic responses, and in a sense this is quite plausible. A movement, of course, is the resultant of a number of muscular contractions, and these in turn grow out of smaller, immediately preceding, contractions, all probably indicative of corresponding neural action. The slower autonomic responses which are associated with the external response likewise have their development, though it is only a hypothesis that they are related to movement responses. A description of the somatic state at the time of an action would give its constituents and would of necessity add up to the action itself. Taken at a time prior to the overt response, the somatic description would undoubtedly correlate with the response; at how early a point there would be such a correlation is a question for experimentation to decide.

Somatic responses will also have their stimuli. In the present report it is their relation to stimuli which is the principal concern of three experiments. All three may be thought of as experiments in a "free response" situation. The subject is told what stimuli he will receive, but is given to understand that no response is required of him. The brief disturbing force (stimulus) is then imposed upon *S* and its effect upon his various response systems is recorded. The free response situation is interesting because it is so common, and seems to provide a great deal of the background of somatic response with which a person lives. The study of this situation should disclose what sort of response tendencies *S* reverts to when no requirements for action are imposed. Such response organizations could be, but need not be, permanently fixed by the structure of the individual. In any case, however, they would be the usual constitution of the individual. Somatic response tendencies under this condition, of course, invite comparison with those that are found when something in the situation provokes an outward movement, possibly an adjustive one.

Three sorts of stimulus variation are introduced in the present experiments. In the first a rather simple auditory stimulus is given repeatedly through a sitting. In the second the principal interest is in the relation of somatic response to stimulus intensity and the possible interaction between intensity and repetition. In the third the stimulus variation is qualitative. Various tactual stimuli were used because some incidental observations suggested that they would have different effects upon *S*.

The recording techniques were chosen so as to give as complete a representation of the somatic responses as could be arranged. One may consider the effect of the experimental variable on each of the responses taken separately. The greater interest, however, is in the pattern of the response when all the recorded measures are considered together; and pattern analysis is notoriously difficult. When records are measured, the tabulations form a manifold of at least four dimensions. There is one dimension to represent the experimental variable (stimulus character in the present case), one to represent individual *S*s, one to represent the time (with reference to a stimulus)

at which a measure was taken, and one to represent the several response variables. In addition, there will be a fifth dimension to represent the position of a stimulus in the series given during a sitting, when that variable is not experimental. Undertaking to find and somehow to quantify patterns in so many dimensions at once would be a hopeless prospect, and one must simplify the problem by selection and combination. The strategy adopted was to study each response variable first as a function of time from the stimulus. This inquiry will detect the presence of a response, its latency, and its course of development. With this information at hand, it is possible to select a single reading for each response variable to represent the magnitude of the response. These values can then be studied as functions of the experimental variables.

For the study of other experimental variables the effects of position are first eliminated by combining all serial positions; then when the experimental design permits, the interaction of an experimental variable and serial position is investigated. The consistency of individuals in their autonomic responses has been examined by Lacey *et al.* (32) and Malmo *et al.* (35), and partly for this reason it is not given primary attention here. The differences between individuals are used, of course, in testing the significance of our experimental effects, but the principal concern will be with how the average individual reacts to stimuli of various kinds.

With these simplifications we finally arrive at tables in two dimensions: one for the experimental variable, in the rows, for example, and one for the response variables, say in the columns. Each row will then specify the response pattern for a particular experimental condition for the average *S*, and the problem will be to find whether the patterns in the several rows differ from one another qualitatively, or perhaps only at random. (The problem of a suitable common metric will, of course, need to be considered.)

For whatever patterns occur, whatever their relations, we shall avoid using such well-worn names as fear, anger, anxiety, etc. To call a response pattern by such a name would be to imply that it has the functional properties assigned to these abstractions in one or another of the widely varied treatments of emotion, psychotherapy, etc. Such an identification would hardly be unambiguous, and at best would be an anticipation of experimental results on what the behavioral relations of the patterns are. We can hardly say, therefore, that we are setting out to discover the physiological basis of anxiety or anything of the sort. We should say rather that our experimental results may bear on the problems for which such concepts have been invented. By establishing that such and such a pattern, perhaps arbitrarily named, is produced in the average individual by one specified stimulus condition and a different one by another, we should hope to establish some landmarks for plotting the terrain. On later occasions, then, as responses are observed under more complex conditions, it would be possible to say, "These are class *xyz* responses such as are typically found in conditions *abc*."

In speaking of classification of response we do not mean to prejudge the continuity of the distributions to be found. It may be that individuals give somatic responses in a limited number of distinct patterns. There is a good chance, however, that nature has not arranged them in such convenient pack-

ages, but allows them to occur in all grades of intensity and a continuous series of patterns. If that should be true, classification would be a crude but possibly useful way of dealing with the data.

II. THE ORGANIZATION OF RESPONSES IN THE CARDIOVASCULAR SYSTEM

The circulatory system, of course, keeps blood flowing continuously to all parts of the body. In the living healthy individual there is a degree of constancy about this circulation: this is true by definition, for should the circulation vary in some manner beyond prescribed limits, the individual is no longer adjudged to be healthy. Much attention has been given to the homeostatic adjustments which help to preserve physical and chemical constancy in the circulation, and some writers (e.g., Ogden 39) see almost any event in the system as maintaining the constancy of something.

But circulatory constancy can be overemphasized. Certainly it is not always achieved, as witness the persons who become "unhealthy" by reason of cardiovascular changes. Even when the derangement is not so severe, there are variations in the system which are probably important in the behavior of the individual. The process called homeostasis, of course, does imply variation. The word seems to have two senses: the adjustment of the system in the presence of a disturbing external force, and the readjustment of the system after the force has been removed. In the first sense it is clear upon reflection that a system which is subject to any disturbing force cannot return itself in all respects to its former state so long as the disturbing force continues. The impinging energy cannot be made to vanish without trace: one feature of the system, such as pressure, must be modified to maintain another, such as constant flow. With thermostatic control a furnace may keep the temperature of a house fairly constant in varying weather, but the action of the furnace itself is far from constant. In the second sense, restoration can be complete, but it is not instantaneous: there will be a wave of activation and recovery, perhaps oscillatory, depending on the time constant and damping parameter of the system.

The fact is (and it is easy to observe) that the cardiovascular system is constantly in a fluctuating condition, with its variations often quite evidently reflecting stimuli outside the individual. Such variations may be of interest to behavioral science.

The homeostatic conception of the circulatory system is too narrow for our purpose. More generally the physiological system which circulates blood can be looked upon as a physical response system which gives an output at a certain point of measurement when an input force impinges upon it somewhere. In a particular case the output in question might be the systolic pressure and the input a fluid put into a vein, or a train of nerve impulses arriving in the muscle fibers of the heart. The output will depend, in general, upon the nature of the input and upon the dynamic structure of the system. This particular system is known to contain some negative feedback loops which under certain conditions will produce homeostatic effects. These effects, as well as those of the other structural characteristics of the system, will appear in one or another of its outputs.

Inasmuch as the output, either to a recording instrument or to the biological mechanisms of the body, is determined by these structural characteristics, it is appropriate to look into what is known or can be discovered about the dynamic organization of the cardiovascular system. This inquiry will bring to light certain technicalities which the student of behavior might prefer to leave buried in the work of a few physiologists

and biophysicists, but for a rational choice of what responses to record and how to interpret them there seems to be no avoiding such details.

As a physical structure the circulatory system is a closed hydraulic loop containing a force pump. (This loop consists, of course, of two subloops, the pulmonary and peripheral, forming a figure eight, as it were, with one-half of the heart in each part.) Flow from the heart is maintained by the force of its beat, but the pressure gradient it produces declines almost to zero in the capillaries, and extrasystemic forces are necessary to return the blood (Ponder, 40, and Green, 19). These are provided in important measure by the compressive action of skeletal muscle contraction and by the negative pressure in the chest cavity which results from respiratory movements. As in hydraulic systems in general there are three physical variables, pressure, flow, and resistance, which describe the system in a steady state. These are related to one another in the same manner as their electrical analogies, e.m.f., current, and resistance, are related by Ohm's law. For a steady state, therefore, any two of the variables would specify the over-all state of the system.

But, of course, the system is not in a steady state: the pulsations introduced by the heart and the periodic responses in which the psychologist is interested continually introduce changes into it. Because of the elasticity of the vessel walls the system is physically reactive to the change, as an electrical circuit containing inductance is reactive. The disturbance will take some time to produce a new steady state, and there will be a definite transmission time from one part of the system to another (King, 30). In a given individual the velocity of this transmission would be expected to vary chiefly with blood pressure, and might serve as an index of pressure variations. This hydraulic conduction is much slower than most nerve conduction, yet much faster than the transport of blood. It will proceed in both directions from the origin of the disturbance except at the heart and in the veins, where the valves prevent any substantial backward transmission.

Forward transmission through the heart is, however, of the greatest importance. It takes place because the force of the beat is principally determined, without neural intervention, by the distension of the ventricles, which itself depends upon the local venous pressure, according to Starling's "law of the heart" (21, 22, and 27).[3] This pressure, in turn, is affected by anything that happens anywhere in the system.

Within the circulatory system there are a number of regulatory mechanisms which operate as feedbacks. Certain of these, operating in a negative fashion, tend to maintain homeostasis; others, with respect to certain variables at least, operate in a positive fashion and tend to exaggerate any disturbance (2). Some of the feedbacks operate altogether by hydraulic transmission; others are neural reflexes.

An important neural regulator is the reflex whose receptors are located in the aortic arch and carotid sinus (23). These receptors are stimulated by pressure in the arteries and are normally in a state of activity. The excitations are carried to the vasodepressor center by the vagus and glossopharyngeal nerves. Efferent impulses (parasympathetic) are then returned to the heart, slowing its rate, and to the walls of the vessels, causing them to dilate. (Perhaps the dilation is brought about by inhibition of sympathetic constrictor action.) The latter effect would lower the pressure and temporarily decrease the amount of blood returned to the heart, thereby acting as a negative feedback with a stabilizing effect. At the cost of a changed state of the nervous system, the circulatory disturbance is reduced, even in the face of a continuing external stimulus. The change in heart rate, however, over the normal range of operation, probably has little effect on mean blood pressure (27). According to Starling's law the greater accumulation of blood during the longer interbeat intervals simply increases the stroke volume of the heart, so that its output per minute would remain the same. This output per minute is, of course, the heart's contribution to mean blood pressure.

The effect of these depressor reflexes is such as to make the heart rate usually an inverse variant of the blood pressure (Marey's law) (27). A slowed pulse is generally associated with an increase in blood pressure. Typically, a high pressure slows the pulse, and the slow pulse (Starling's law) results in larger stroke volume and pulse pressure.

A similar pressure reflex operates from receptors in the large veins near the heart over a vagus afferent and a sympathetic efferent. When venous pressure rises, this reflex produces cardiac acceleration (Bainbridge reflex) and possibly vasoconstriction (MacDowell reflex) (21, 2). The latter therefore operates as a feedback with a positive sign, increased venous pressure leading to events which produce a further increase in venous pressure. While this effect is the opposite of homeostatic for the circulatory system, it may well be homeostatic for inter- or intracellular fluids. The Bainbridge reflex has been interpreted as a mechanism to prevent the heart from receiving too great a charge of blood on a single

[3] Certain investigators (45) have questioned whether Starling's law adequately accounts for the action of the heart in the intact individual. Their evidence, however, has not been sufficient to convince most physiologists.

beat (39). Its relations to the aortic arch reflexes seem not well understood. On the one hand, the aortic arch reflexes seem to be dominant—in the long run they or their equivalent must be, or there would be no stopping. On the other hand, there are (during some kinds of stimulation) occasions when heart rate and vasoconstriction are maintained at an elevated level.

If we wish to consider the cardiovascular organization as a response system, we must identify its input and output. From the standpoint of the organism's economy the output of the system would be the substances which it discharges at various points in the body. In the final accounting these may be the important outputs for behavioral studies also. It is possible experimentally, however, to take as an output any variable aspect of the system which can be recorded, and for analytic purposes an event earlier in the chain would often be a good choice. The physical properties of the system would be classed as pressure, flow, and resistance variables, and each class would have many varieties. Choice would be affected, of course, by knowledge of their relationships.

Inputs to the system may be mechanical or chemical, coming from other organ systems, but for immediate adjustments the most important inputs are undoubtedly neural. These provide a coupling of the system to the other parts of the body and, through the exteroceptors, with the events outside the organism. Neural inputs act at two locations in the cardiovascular system: the heart and the small peripheral vessels. (Arteries and veins are little affected by neural action.) If the force of the heart beat is entirely regulated by Starling's law, the effect of neural impulses on the heart would be to modify the rate at which the heart beats and its stroke volume, the latter indirectly and inversely. Peripherally, the neural effect would be to contract or dilate the small vessels through the action of the smooth muscles in their walls.

At each of these sites the effect may be in either positive or negative direction, and either direction may be produced by direct- or inverse-acting nerve fibers (for the heart, pressor and depressor; for the vessels, constrictor and dilator). An increase in the action of one of these will be equivalent in sign to a decrease in the action of the others. For the heart the direct-acting nerve (pressor) fibers are sympathetic, the inverse-acting ones, parasympathetic. For the vessel walls the direct-acting fibers (constrictor) are sympathetic. On the other hand the inverse (dilator) fibers are parasympathetic and sympathetic (by origin at least) and somatic (2). These fibers conduct the curious antidromic axone reflexes which produce vasodilation primarily at the site of stimulation.

The neural pathways from external sense organs to the cardiovascular system are quite numerous. The simplest is the already mentioned axone reflex in which afferent nerves conduct in both directions to produce vasodilation. More complex routes for both direct- and inverse-acting systems probably lie through the spinal cord, through the medulla, and, superimposed on these, through the midbrain and the cerebral hemispheres. For the vasoconstrictor effects, it is said that stimulation of any of the afferent nerves (except those involved in the carotid sinus-aortic arch reflexes) will produce them without necessarily having any direct effect on the heart (2). The action of the sympathetic in constricting peripheral vessels is usually thought to be spread through the whole organism. Its effects, however, are not the same in all regions. The vasculature of the brain, for example, though innervated by the sympathetic, is little subject to the vasoconstrictor reaction (44). Consequently, a general vasoconstriction will have the general effect of increasing the blood flow through the brain. In muscular and glandular regions the vasoconstriction is also differential because the local dilation (produced by the metabolites of local activities) is prepotent over the vasoconstriction (23). Constriction in the less active parts, therefore, will force more blood into these active parts. Vasodilation is reported to be produced by the stimulation of certain local regions, for example, parts of the sex organs. Presumably the pathways for any of these, as for pressor and depressor effects on the heart, would be through the autonomic centers by means of side branches from ascending tracts or by a return from the cerebral hemispheres through the autonomic centers.

The possibilty of understanding a train of cardiovascular events by means of the known system characteristics may be tested by examining some recorded responses. Two studies to be analyzed in other ways in the following sections provide two sets of data which are appropriate to the purpose. In both of these studies a variety of circulatory responses as well as certain others were recorded while *S* was being given loud tones of two-seconds duration at intervals of one minute. The primary purpose of these studies, as set forth later, with further experimental details, was to test the effects of repetition in the first, and of stimulus intensity in the second. A special set of measurements was made

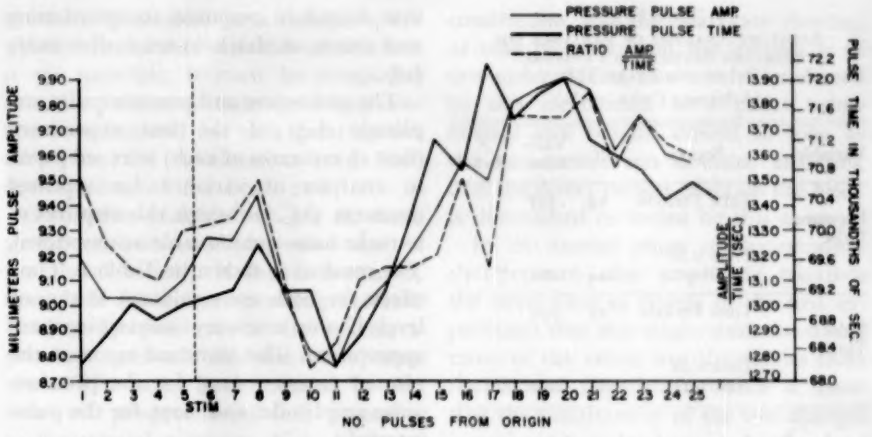

FIG. 1. Effects of auditory stimulus on pressure-pulse amplitude, interpulse interval, and their ratio. (Tone 98 db., 2-sec. duration, begins at line marked "stim." Means of 17 *Ss*.)

on the records to provide information on the relations between circulatory events. Our immediate interest is in pressure pulse, volume pulse, and interpulse interval following the stimuli.

For interpulse interval and size of arterial pressure pulse, measurements were made on each of 5 pulse beats before each stimulus onset and on the 20 following pulses for the first group. For this group the volume-pulse amplitude was measured by dividing the record into two-and-a-half-second periods and measuring the maximum amplitude in each for two periods before the stimulus and four periods following. The amplitude measurements for the pressure pulse are in arbitrary units. In recording, the practice was to adjust gain controls so that each *S*'s resting pulse was of about the same size. If a substantial change occurred in the resting state pulse, the gain controls were readjusted and appropriate correction made in the measured amplitude. In the case of volume pulse, which was measured at specified times, the amplitude measurements were expressed as percentage of each individual's prestimulus mean reading prior to each stimulus. These conventions are somewhat arbitrary, but probably could be varied considerably without much change in the results.

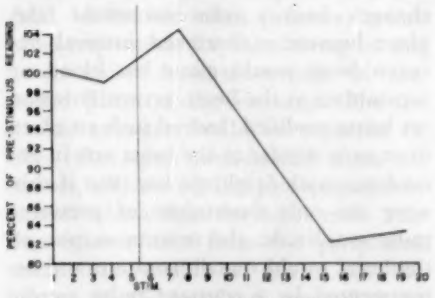

FIG. 2. Effects of auditory stimulus on volume pulse. (Recorded simultaneously with Fig. 1 data.)

The means of the values so obtained for the first (repetition) experiment are shown in Fig. 1 and 2. In order that readings in Fig. 2 correspond in time with those of Fig. 1, the abscissa scale has been adjusted by the pulse-time data so that all pulse times are represented by equal intervals as they are in Fig. 1.

TABLE 1
AUDITORY ANALYSIS OF VARIANCE FOR STIMULUS EFFECTS ON PRESSURE PULSE AND PULSE TIME
(Arbitrary Units, 16 *Ss*)

Variable	Source	*df*	Variance	*F* Ratio
Pressure Pulse	Time Periods	14	.597	
	Time × *Ss* (Error)	224	.263	**2.7
Pulse Time	Time Periods	14	.032	
	Time × *Ss* (Error)	224	.010	**3.2

(This physiological time unit seemed appropriate to the data.)

For the second experiment (intensity) the data were treated in much the same fashion, but are not presented here because they are substantially identical in character.

In both experiments it is evident that pulse time and pressure-pulse amplitude show a diphasic response, becoming smaller and faster with a minimum amplitude and time at 5 to 7 beats after the stimulus onset, then increasing in size and slowing in rate, so that both time and amplitude exceed those of the prestimulus level. In the second experiment the deceleration has in turn disappeared by the 20th poststimulus beat, while the increased amplitude still persists. A similar situation may be developing in the first experiment, where measurements were not extended so far into the poststimulus period. Meanwhile, the peripheral vessels (Fig. 2) are undergoing a single phase response of vasoconstriction, beginning about the 3rd poststimulus beat, reaching its maximum at about the 10th poststimulus beat, and then slowly recovering. The behavior of these variables is much like that found in response to questioning and threat of shock in an earlier study (16).

The pulse-time and pressure-pulse amplitude data of the first experiment (first 15 measures of each) were subjected to analyses of variance for repeated measures (15) (although this requires us to make some questionable assumptions). The results are shown in Table 1. Time effects for both are significant at the .01 level if the necessary assumptions are appropriate. The standard error of the plotted means is .125 for the pressure-pulse amplitude, and .0075 for the pulse interval.

Returning now to a consideration of the circulatory mechanisms, the initial acceleration of the pulse and the vasoconstriction which seems to develop somewhat later, indicate effect of the sympathetic division on the two input points—heart and peripheral vasculature. This effect would be expected from the report (2) that stimulation of any afferent nerve brings about sympathetic excitation.

Changes in pulse time and pressure-pulse amplitude are remarkably similar. They are so much alike that it seems at first that the pulse amplitude change may be merely a reflection of the rate changes. Such a reflection would take place because a shortened interval between beats would allow less blood to accumulate at the heart, return flow not yet being modified. Indeed such an effect must exist insofar as the heart acts in accordance with Starling's law. But if this were the only determiner of pressure-pulse amplitude, the minute output of the heart would remain constant and be represented by a constant pulse amplitude per unit of time. Such an index was therefore computed, and the plot of it is shown in Fig. 1. It is clearly not a

constant, but varies in a fashion quite parallel to the two variables of which it it the quotient. It must be concluded that the pulse-amplitude variations cannot be accounted for to a great extent simply by the acceleration (and later deceleration) of the heart rate.

The other possible variable is, then, peripheral resistance (decreased size of peripheral vessels). If this is increased, there will be a corresponding increase in diastolic pressure in the system, a pressure against which the heart must work. The same force exerted by the heart will produce less of that increment in the arterial pressure which shows up as the pressure pulse. We have independent evidence from the volume-pulse record that there has been this peripheral vasoconstriction. This would produce the increased resistance and increased diastolic pressure, which would produce the diminished pressure pulse. In the first phase of the response, therefore, there is an acceleration of the heart, and a peripheral vasoconstriction, which shows up as diminished volume and pressure pulse. If this analysis is correct, there is an increase of diastolic pressure during this period, but no change in systolic.

The increase in diastolic pressure (and, of course, mean pressure) seems to be responsible for the slowing of the heart which is characteristic of the second phase of the response. The increased pressure would be expected to affect the aortic arch-carotid sinus receptors, and set off the parasympathetic reflexes which slow the heart rate. The transmission time from the peripheral vasculature would be small, but the latency of these reflexes, according to studies of Matthes and Ebeling (36) is from one to two seconds, depending on the individual. The lag between vasoconstriction and the heart-rate slowing, as may be seen from the graphs, is of this order. Heart rate would be slowed by this mechanism to a point below normal and remain slowed so long as the vasoconstriction obtains, assuming that the direct pressor effect on the heart is diminished or ended by this time.

In the second phase of the response the pressure-pulse amplitude parallels the heart time so closely in the first experiment that one might seem the direct cause of the other, but the results from the second experiment make it clear that the simultaneity of the two changes is not necessary. In these results the slowing of the pulse definitely precedes the increase in pressure-pulse amplitude.

This effect seems to derive from peripheral constriction, operating in a forward rather than a backward direction in the circulatory system. Vasoconstriction of the peripheral vessels will force most of their contents forward through the veins to the heart. Venous pressure will be increased and blood will accumulate more rapidly at the heart. Hence even for a fixed interbeat interval, the heart will receive a greater charge of blood and in accord with the Starling law respond with a stronger beat. More blood, at higher systolic pressure, will be ejected into the arteries and cause an increased pressure pulse. It is in this phase of the response, therefore, that we should find an increased systolic pressure. This would be in accord with Marey's law: high systolic pressure and slow pulse would be associated. Some time would be taken for the effects of vasoconstriction to reach the heart by means of this hydraulic transmission and the graph shows that such is the case.

The data of the two experiments are therefore related, as one would expect

in the light of the dynamic structure of the response system on the assumption of the predominantly sympathetic type of initial response arising from the auditory stimulation. Data from the other circulatory variables, reported in the following sections, generally fit into this interpretation quite well and sometimes reveal further details of its operation.

It may be convenient to speak of a sequence of events such as that found in these two experiments as a Type 1 cardiovascular response. In summary, it is characterized by a first phase of cardiac acceleration, vasoconstriction, smaller pressure pulse and probably increased diastolic pressure. The second phase is characterized by continued vasoconstriction (with some recovery), stronger pressure pulse, subnormal heart rate, and probably increased systolic as well as diastolic pressure. The peripheral constriction of the first phase seems to bring on the second phase by acting in two directions: by increasing mean arterial pressure it activates the heart-slowing reflexes, and by increasing venous pressure and flow it returns more blood to the heart and increases the force of its beat and the amount of blood ejected.

The associations noted in the cycle lead to certain guiding principles for the selection of recording techniques. When a Type 1 response is to be recorded, it is clear that the same mean result will be obtained from pulse time and pressure-pulse amplitude up to the point of response maximum. Between the two the choice would be on the basis of convenience only. A good cardiotachometer would probably make the rate measure the more convenient. The relation of time measure to c.g.s. units is easier to determine than is the relation of such pressure measurements as here used. Because of the important role played by peripheral vasoconstriction, there would be experimental problems for which volume pulse would be a specially valuable measure. In the chain of events it is close to the neural input, and its consequences are of major importance for the circulation, at least. Vasoconstriction is, to be sure, a local condition; it may or may not occur in other peripheral locations at the same time. So, if a single measure of total systemic effect is desired, volume pulse at one site would be untrustworthy. But if there is interest in neural action at various sites, and in the consequent redistribution of blood, the volume measure taken at several sites would be most informative. It might be added that a convenient feature of the volume-pulse response is its monophasic character, which makes it easier to quantify.

The analysis of the data shows that the circulatory mechanism can be treated as a physical response system, and that the diverse manifestations of change within it are related to each other as would be expected from the dynamic characteristics of the system. The case considered is, of course, the impressment of a temporary disturbing force (a "square wave" at the external receptors). This input, or stimulus, initiates a train of events in a rather slow-acting system, much as a sharp push would start a movement in a heavy pendulum, a movement which reaches its greatest excursion long after the applied force has ceased, and which spends itself after a still longer time. Of course, events in the circulatory system are more complex, and as the response progresses it changes character by more than simple oscillation. There are negative feedback mechanisms in the system: for example, the aortic-arch carotid sinus receptors initiated a vasodilator

effect when they are stimulated by pressure. During the response these mechanisms would produce a slowing and braking effect, and they would operate to return the system to its original state sooner than it would otherwise return. These homeostatic devices would modify the response, but they evidently do not prevent it, nor could they.

Different inputs to the circulatory system would undoubtedly produce different responses. These will be molded, as it were, by the dynamic characteristics of the system. Recordings of the several output variables under other conditions will probably show them in new relations and new sequences. The large body of data coming from such studies will need to be organized by some means: analytic knowledge of the response system should prove a valuable basis of interpretation —perhaps a necessary one. The Type 1 response here described is possibly only the beginning of a series of types, and with the identification of this one it should be possible to discover under what other external conditions it will occur, and under what conditions there will be departure from it.

III. THE EFFECTS OF STIMULUS REPETITION

In any program of researches designed to discover variables which determine the magnitude and interrelationship of autonomic responses, the effects of repeated stimulation must command a high priority. Besides being important in its own right, the repetition effect is fundamental to the study of most other independent variables, inasmuch as experiments will usually require the presentation of more than one stimulus to each *S*.

That the galvanic skin response diminishes with repeated elicitation is well known (52), but apparently little is known about other autonomic responses in this regard. The GSR effect may be due to local conditions, such as exhaustion of the sweat glands, or it may be characteristic of the autonomic system or of the whole response mechanism. If either of the latter is the case, other autonomic responses should show a similar extinction effect. A simultaneous study of the effects of repeated stimulation on a number of autonomic responses should show whether GSR extinction is indicative of a general systemic effect or is merely a local obstacle to discovering the state of the autonomic system.

An earlier series of studies (16) contained indications that some of the autonomic responses studied were subject to the adaptation effect, while others did not appear to be. Several studies of the effect of stimulus repetition on the tension response in skeletal muscles indicate that fairly rapid adaptation occurs unless responses are followed by what amounts to a sort of reinforcement (9).

The present study is designed primarily to compare extinction effects in several autonomic responses. Put another way, it will examine the constancy or variation of the autonomic response pattern under repeated stimulation. If one kind of response adapts readily and another does not, there is, of course, a change of response pattern.

A. Method

Apparatus. Autonomic responses were recorded with elements of the apparatus described earlier (10, 16). The sound source was an oscillator which was set to deliver automatically a rough tone of 98 db. and approximately 800 cycles, with a duration of 2 seconds,

once a minute. *Ss* were seated in a shielded room and received stimulation over a speaker system.

Subjects. *Ss* were 17 undergraduate students recruited from elementary psychology classes.

Procedure. The *S* was seated in the shielded room and attached to the various devices (see below) which were connected to the recording apparatus in the next room. He was instructed that sound stimuli would be given periodically and that he was to do nothing but remain as quiet as possible. After about 5 minutes of rest during which apparatus adjustments were made in the recording room, the motor which governed the automatic presentation of stimuli was started, and allowed to run until 10 stimuli had been presented. During this period continuous recordings were made of the following variables:

1. Skin resistance, from the left palm
2. Pressure pulse—left radial artery
3. Chest breathing
4. Blood pressure (systolic)
5. Volume of tip of left index finger
6. Volume pulse from tip of left index finger

Pulse and breathing yielded both amplitude and time-of-cycle measures; GSR and finger volume yielded amplitude and latency measures.

B. Results

The recordings make possible a study of repetition effect on the following ten variables: GSR magnitude, GSR latency, pressure-pulse amplitude, interpulse interval, breathing amplitude, breathing time, systolic blood pressure, finger volume, finger-volume response latency, and volume-pulse magnitude. The nature of the measurements made on each variable and of the results obtained will be discussed below.

All amplitude measurements were adjusted according to the sensitivity setting of the recording device at the time the record was taken.

The problem of adequate statistical treatment of results looms large in the present experiment owing to the difficulties involved in simultaneous treatment of data based on repeated measurements. The various proposed solutions (1) involve assumptions of normality of distribution of variables and homogeneity of variance or of parallel regression lines between individuals. Because it appeared to be extremely unlikely that any of the dependent variables would justify these assumptions, analysis-of-variance tests were not used. Instead, the presence of a repetition effect in the data was tested in several different ways, and the technique described below was selected for presentation because, with one exception, it enables rejection of the null hypothesis for every variable for which any other test enables rejection of the null hypothesis at the .05 level of confidence.

For each variable separately, the mean response to each stimulus occurrence was calculated. The means were then ranked from largest to smallest response and the ranks summed for the first five stimulus occurrences. The probability of obtaining various sums of ranks was easily computed by summing various combinations of numbers and dividing the number of combinations equal to or less than each possible sum by the total possible number of combinations.

The null hypothesis tested is that the size of mean response is independent of stimulus order. If this is the case, the sum of ranks for trials 1 through 5 would be expected to be 27.5 (half of the sum of numbers from 1 through 10). For variables where the sum of ranks of trials 1 through 5 is extremely low or high it can be concluded that the order of the mean responses is not random and that there is probably an effect of repeated stimulation. (This test is indirectly affected by increased number of *Ss*, since an increase in sample size yields better estimates of population means.)

Table 2 presents the sums of ranks for the dependent variables.

1. Skin Resistance

Galvanic Skin Response (GSR) Magnitude

A reading was taken of *(a)* the minimum resistance within the 10-second

period following each stimulus occurrence, and *(b)* the resistance level just prior to each stimulus occurrence (base level). Each of these was then translated into ohms using calibration constants determined after each *S* was run. Data from an earlier series of experiments (16) indicated that the means and standard deviations of the responses at various base levels were nearly linear functions of the base level and that the regression lines passed through the origin. A data plot from this experiment shows a somewhat similar relationship with the possible exception of a downward drop for very high base-line values. (These however involve only a few measures.) Under these circumstances, if we take as a GSR measure the ratio a/b (i.e., decrease in resistance divided by base level), means and standard deviations for varying base levels should be essentially constant and consequently independent of the base level.

In our data there is a rank-difference correlation of — .81 between mean base level and serial position. Since there is also a negative correlation between the mean GSR and serial position, it follows that there must be a correlation between the serial-position means for the two variables (base level and GSR). This actually amounts to .62. The significant fact for our purposes, nevertheless, is the noncorrelation of the ratio and the base level, with serial position constant.

Accordingly, we have used a measure of GSR magnitude given by the expression, response/base line × 100. Figure 3 shows the means of this measure for all *S*s and for each stimulus occurrence expressed as a percentage of the largest mean value, with these values plotted as a function of stimulus occurrence. In this experiment the mean value for the 10th stimulus occurrence is about 15% of that of the first. Examination of Table 2 leads to the conclusion that the decline in GSR magnitude seen here is unlikely to be a random fluctuation. In a way, this sets the principal problem for the study of the other response variables: will they behave in a similar fashion?

TABLE 2
SUM OF RANKS FOR TRIALS 1–5 FOR RESPONSE VARIABLES

Variable	Sum of Ranks
GSR	17*
GSR-latency (reciprocal)	24
Pressure pulse	23
Breathing amplitude	39**
Breathing cycle time	21.5
Interpulse interval	17*
Systolic pressure	25
Finger volume response	22
Finger volume latency (reciprocal)	26
Volume pulse	17*

* .03 level of confidence.
** .016 level of confidence.
"Sum of ranks" has a theoretical maximum of 40, minimum of 15, mean of 27.5.

GSR Latency

Measurements were made of the time from stimulus onset to the beginning of each subsequent decrease in resistance. In 37 of 170 cases no decrease could be noticed, and therefore no measurement was possible.

With no transformation the distribution of latencies, for all cases in which responses occur, is J shaped. Not only is this distribution inconvenient, but there is also no way of representing quantitatively instances where no response occurred. To overcome these problems latency measures were subjected to a reciprocal transformation. This allows us to assign a value of zero to instances in which a response did not occur, and, with the exception of a piling up of measures at zero, results in a more symmetrical distribution of measures than

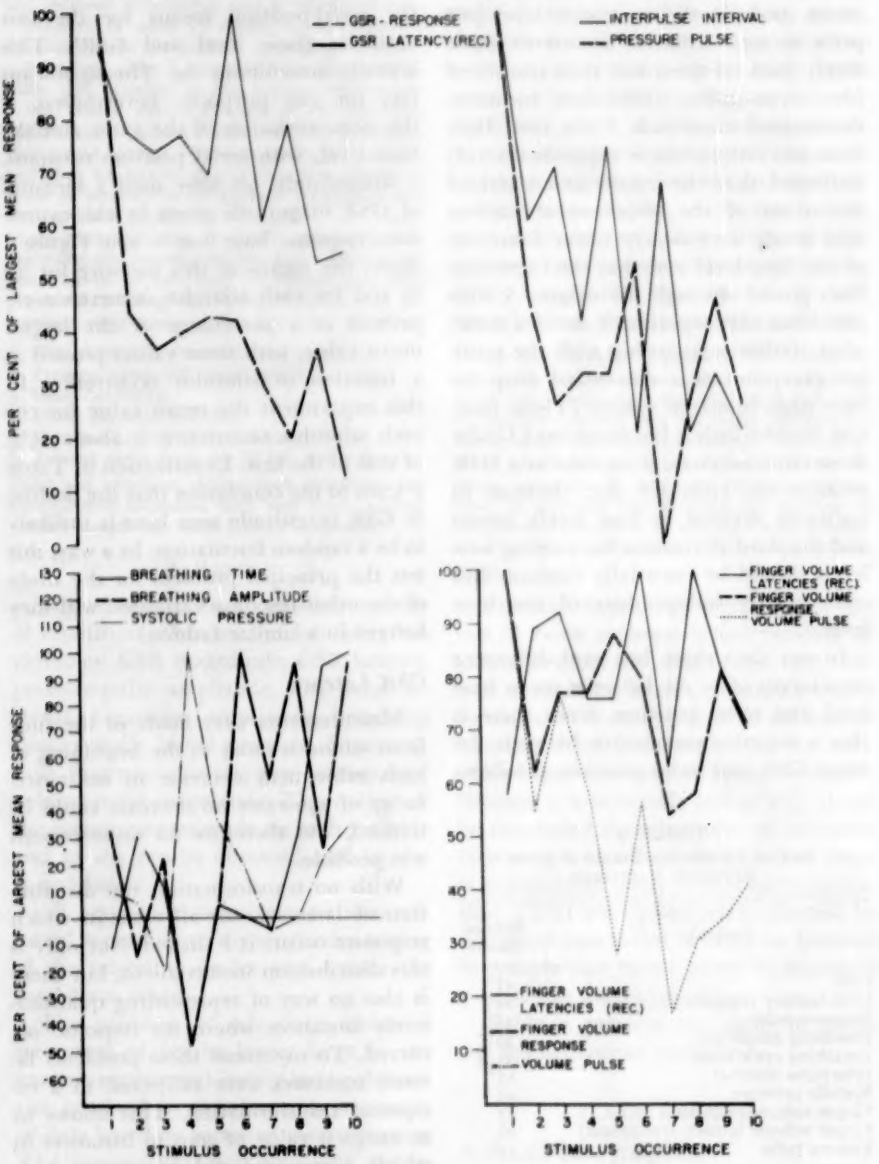

FIG. 3. Size of response in the several variables to repeated presentations of a 98-db. rough tone of approximately 800 cycles.

was the case prior to transformation. Figure 3 shows the means of this measure for each stimulus occurrence.

A problem arises in comparing GSR latency measures over a series of repeated trials. As the number of stimulus occurrences increases, the number of individuals who do *not* show a GSR to the stimulus increases, i.e., not only magnitude but also frequency of response decreases with repeated elicitation. Assigning a value of zero to latency reciprocals for nonoccurring responses thus tends to *increase* latency as stimulus occurrences increase. The means of the reciprocals for each stimulus occurrence expressed as percentage of the largest mean and plotted against stimulus occurrence in Fig. 3 show this downward tendency. While the sum-of-ranks test does not allow rejection of the null hypothesis for this variable (Table 2), a *t* test between paired first and last measures for each individual *is* significant at the .05 level of confidence. But this result is (at least partly) a function of the greater number of failures to respond to the tenth stimulus occurrence than to the first stimulus occurrence. The mean reciprocals for each stimulus occurrence based only on the number of *Ss* showing a response give results in the same direction, i.e., an increase in latency, but again the sum-of-ranks test does not allow the rejection of the null hypothesis.

2. *The Pressure Pulse*

The measurement of the pressure pulse was accomplished by dividing the record into five-second intervals, one before and four after *each stimulus occurrence*. These are designated as B, A_1, A_2, A_3, and A_4. The maximum amplitude change of the pulse, i.e., the difference between the highest and lowest excursion, was ascertained for each such interval.

To adjust for individual differences in level, which are chiefly due to arm structure and manner of attachment of the instrument, we determined the mean of the 10 B intervals (one for each stimulus occurrence) for each *S* separately and then converted all readings for any *S* to percentages of his mean B reading. For each of the five intervals (B, A_1, A_2, A_3, A_4) the mean of these percentages was computed (summing over all stimulus occurrences for all *Ss*). These means were then examined for the presence of a stimulus effect. From the mean B reading there is an increase, with its maximum of about 6% between 5 and 10 seconds after the stimulus onset. This is in general accord with the results of the beat-by-beat analysis reported in an earlier section.

To make the comparison of responses to the successive stimuli, a response was defined as the difference between the reading in the period between 5 and 10 seconds after stimulus onset (A_2) and the reading just before stimulus onset (B). The mean response to each stimulus was calculated and expressed as a percentage of the mean response to the largest stimulus, which in this case is the first. Figure 3 shows these values.

Although the response to S_{10} is less than 30% of that to S_1, the sum of ranks does not depart significantly from a chance level. The decrease in response found here cannot, therefore, be claimed to be more than a random fluctuation.

3. *Chest Breathing*

Amplitude

Five-second intervals similar to those described for pressure pulse were used. In each interval the difference between maximum and minimum excursion was measured and the mean difference ob-

tained (summing over all *Ss* and all stimulus occurrences). Examination of these means shows an increase in the amplitude of breathing occurring following stimulation. This reaches its maximum as late as 15 to 20 seconds (A_4), after the stimulus where there is a mean increase of 16% over prestimulus level (B).

When means of all *Ss*' readings were computed for each interval for each stimulus occurrence separately, a surprising relation appeared. Product-moment correlations between the mean for any time interval and the difference between it and the mean of the next time interval were negative and fairly high (median $r = -.64$). Examination proved that this was a mathematical consequence of substantially zero correlation between the means of readings taken in the various five-second intervals, that is to say, knowing the mean breathing amplitude in one interval enables one to make no prediction of the relative mean amplitude in the succeeding interval. In such a case the differences between time intervals will be correlated with the differences between the next pair of time intervals to the extent of $-.70$. Our high negative coefficients therefore merely indicate that the component measures are uncorrelated. This is a relation which invites further study, perhaps by autocorrelation (serial correlation) (25), for it is quite contrary to the impression one gets from individual records.

The lack of correlation of successive means makes it difficult to compare stimulus effects, since one is applying an experimental variable to a magnitude that is constantly increasing and decreasing "spontaneously." There would be little point in subtracting a prestimulus (B) value from the poststimulus (A_4) reading, for one might as well subtract the numbers in the series at random. Accordingly, we have utilized the poststimulus (A_4) readings minus the mean B readings (summed over all *Ss* and all stimulus occurrences). Figure 3 shows the results converted into percentages of the largest "response." With considerable irregularity, there is a strong suggestion that the amplitude is increasing as the sitting progresses, with the response possibly being more delayed.

The results of our statistical tests enable us to conclude that means of responses to the last five stimuli are not drawn from the same population as means for responses to the first five stimuli. But this does *not* indicate adaptation, since the means for the last half of the series tend to be higher than those for the first half. In fact, with breathing amplitude we seem to have facilitation of response with a repeated stimulus rather than habituation of response.

Duration of Breathing Cycle (Breathing Cycle Time)

The same time intervals mentioned under breathing amplitude were used as a basis for breathing-cycle time measurements. The duration of the first breathing cycle in each five-second period was measured. The means of these measures (summed over all *Ss* and all stimulus occurrences) show an increase in time per cycle with the greatest effect (only 2% of the base rate, however) showing in the period 15 to 20 seconds (A_4) after the stimulus.

As with breathing amplitude there is a general lack of correlation between successive mean readings of the breathing cycle duration, and a negative correlation (median $r = -.59$) between the mean level in a given time interval and the gain to the subsequent time interval.

The readings were therefore given the

same treatment as those for amplitude, the mean B reading (summed over all *Ss* and all stimulus occurrences) being subtracted from the poststimulus (A_4) means, and the results converted into percentages of the largest "response." Results are shown in Fig. 3.

Here also the tendency is for an increase in response with repeated stimulation, although none of our statistical tests allows us to conclude that this is more than random fluctuation.

4. *Interpulse Interval*

Interpulse interval is measured in preference to pulse rate because of its greater convenience. The measurement is most conveniently taken from the pressure-pulse record. Five periods, of 2.5 seconds duration, were laid off, one before and four immediately after stimulus onset. These will be referred to as B, A_1, A_2, A_3, A_4, respectively. For each interval the duration of three cycles was measured.

When mean durations (summed over all *Ss* and all stimulus occurrences) are computed for each interval and then expressed as a percentage of mean B measure, the figures are 100.3, 99.8, 101.5, and 101.2 for the four poststimulus intervals in that order. While the increase is slight, even for the third interval, it is apparently consistent. The A_3 interval has the largest value of any of the five intervals for each of the 10 stimulus occurrences.

As is the case with the two breathing measures, there is a lack of correlation between successive measures, making subtraction of prestimulus measures of dubious value.

Accordingly, the response measure chosen was the value for the period from 5 to 7.5 seconds after stimulus onset (A_3) minus the prestimulus (B) mean (summed over all stimulus occurrences). The means for the various stimulus occurrences, in per cent of greatest mean, are shown in Fig. 3. The increase in interpulse interval in response to the tenth stimulus occurrence can be seen as only about 20% as great as to the first stimulus occurrence.

The decrease in response size is significant at the .03 level of confidence. It can be concluded that stimulus repetition serves to decrease the slowing of the pulse which occurs in response to an auditory stimulus.

5. *The Systolic Pressure*

The technique used for measuring blood pressure produced readings only at 20-second intervals. As a preliminary analysis, the first two readings after the stimulus were expressed as percentages of the reading immediately preceding stimulus onset (B). The first value after the stimulus is 4.2% greater than the prestimulus level, while the second poststimulus reading is 2.0% greater than the prestimulus level. There is a great deal of variability in the response: about 41% of the first poststimulus readings are in the opposite direction, i.e., smaller than the prestimulus readings.

The time between the stimulus and the systolic pressure reading was variable from *S* to *S*, and this may account for some of the variability of the readings. (For each stimulus, however, the mean time for all *Ss* was nearly the same.) The rather small size of the response is similar to that found for nonlie questions (16). A study by Darrow (5) also showed little blood pressure change with the type of stimulus used here.

In attempting to define a measure of this variable to be used in the study of repetition effects, the same condition was encountered as was found with the two breathing measures—a lack of corre-

lation between successive measures. Accordingly, we again utilize, for systolic pressure, the first poststimulus reading minus the mean prestimulus B reading (summed over all *S*s and all stimulus occurrences). Mean values of this measure for each stimulus occurrence as a percentage of largest response are plotted in Fig. 3.

The results of statistical tests do not indicate that the variations in mean response magnitude which occur are other than random. Experimental precision for this variable is rather poor, however, for reasons already given.

6. *Finger Volume*

Finger Volume Response

The direct coupled recording from the plethysmograph gives a record that shows, after a certain latency, a wave of constriction. The measurement made for amplitude of response was the amount of decrease from the level prior to onset of each stimulus occurrence to the maximum constriction following the stimulus. After being adjusted for calibration, the means of these values by stimuli were computed. Converted to percentages of maximum response they are shown in Fig. 3. The response is usually present: there are never more than 30% zero responses to a stimulus. The results of the various statistical tests do not indicate the presence of a stimulus repetition effect in the underlying population, although there is a small decrease in the size of the response here.

Finger Volume Latency

Latencies were measured from the beginning of the stimulus to the beginning of the succeeding decrease in volume. A frequency distribution of these measures forms a very regular J pattern. The times reach such lengths that it seemed questionable whether some of the later responses might be spontaneous, i.e., not directly related to the stimulus. As one check on this possibility, measurements were made in a backward direction from the stimulus to the beginning of the first preceding "response," with the thought that the distribution of such measures would indicate the amount of such spontaneous responding. The distribution of these latencies indicates that there is some concentration of responses in the half minute or so before the stimulus. As the delivery of the stimuli was at regular one-minute intervals, it seems likely that some anticipatory responses developed as the sitting progressed. Future experimental design should take some account of the possibility.

Comparing the prestimulus and poststimulus distributions, it seems that the effect of the stimulus is to give a definite J form to the figure, exaggerating the number of short latencies and depressing the number of long ones.

Because of the cases where there is no response (38 of 170), it is desirable to convert latencies to reciprocals, so that the no-response cases may be averaged with the others. When these reciprocals are arranged in a distribution, the form is still of the J type, differing from the outcome with the GSR latencies. The mean reciprocal corresponds to 6.2 seconds in contrast to the mean reciprocal for GSR, which corresponds to 2.5 seconds. The finger volume response quite definitely has a longer latency than the GSR as indicated by simple inspection of the records. The GSR could not, evidently, be influenced by the volume changes (vasoconstriction) as has sometimes been supposed.

Mean values of the reciprocal latency of response to each stimulus expressed

as a percentage of the greatest mean are shown in Fig. 3. No stimulus repetition effect appears.

7. *The Volume Pulse*

Differences between maximum and minimum excursions for two 2.5-seconds periods before the stimulus and four such periods after the stimulus were measured. These will be referred to as B_1, B_2, A_1, A_2, A_3 and A_4, respectively. Since the units were arbitrary and presumably varied from one *S* to another, the measurements were converted by expressing each *S*'s measurements as a percentage of his mean B_1 value.

The over-all means in percentage for the time intervals are 100.0 and 99.0 for the prestimulus, and 104.4, 91.3, 82.2, 83.3, for the four poststimulus intervals in that order.

The expected decrease in the volume pulse, reflecting vasoconstriction, is at its greatest in the A_3 interval measure (5 to 7.5 seconds after stimulus). It is uncertain whether there is a slight preliminary vasodilation as suggested by poststimulus reading. Vasoconstriction is evident in the period 2.5 to 5 seconds poststimulus (A_2).

For the study of repetition effects, the response was defined as the mean of readings A_3 and A_4 minus B_1 [i.e., $\frac{1}{2}(A_3 + A_4) - B_1$]. These differences, expressed as percentage of the largest response, are shown in Fig. 3.

The data indicate that the stimulus has less effect after repeated stimulation. The null hypothesis can be rejected at the .03 level of confidence. It can be concluded that the decrease in magnitude of the volume pulse to an auditory stimulus is less after a series of such stimuli than it is to the first stimulus of the series.

The relation between the volume-pulse effect and the finger constriction effect needs elucidation. It has been supposed that these techniques are two methods of recording the same effect: vasoconstriction in the site measured. Both (it may be recalled) are taken from the same electrodes. Yet there are here shown to be some notable differences between the effects produced by stimuli in the two variables.

At first glance the latency of the two effects seems different, for in the volume pulse there is a substantial change before five seconds poststimulus and latencies for the finger constriction run to very large values. The difference might arise from different methods of measurement in the two cases, but results of the "intensity" study (see below) indicate that the difference is a real one. Further differences are in the amounts of extinction found in the two measures, and in the greater variability in general found in the mean volume-pulse responses. Here again a different method of quantification might produce different results. Nevertheless one should not overlook the fact that the functions may be different physiologically. In measuring the finger volume one gets presumably a value which is proportional to the cross-sectional area of the blood vessels. In the volume pulse one measures the reaction of these vessel walls to a series of impacts, the pulse pressure increments, which, for simplicity at least, one may suppose to be fairly constant over the period concerned. The amount by which the walls are enlarged under these pressures need not be a linear inverse function of their momentary cross-sectional area. Indeed it is not: the resistance offered by the wall of a vessel is a higher power function of its state of distention (19). In the volume pulse we have a measure of this resistance to distention and

volume pulse should therefore vary in the same direction as the finger volume, but in a more extreme manner in accordance with the power function.

C. Comparable Data on Skeletal Muscle Response

The present experiment includes no data on the responses of the skeletal muscles. However, results from a closely comparable experiment on this variable are available (9). In that experiment 16 *Ss* were given 0.2 seconds high-level auditory stimuli at one-minute intervals. The stimuli were white noise at 100, 110, 115, and 117 db. These were presented to the *Ss* in various orders so that each intensity appeared equally often in each serial position. *Ss* were told they were to do nothing in response to the stimuli. The response measured was the increment in log action potentials produced by the stimulus during the period 0.2 to 0.7 seconds after onset, potentials being measured in the flexor and extensor muscle regions of the right and left forearms.

A pronounced extinction of the response was found so that by the 12th trial the response in log terms is only 12% of what it was on the first four. (In raw measures, of course, the difference is even greater.)

D. The Pattern of Response

The autonomic variables which were recorded and the effects of the stimulus at the beginning of the stimulus series are summarized in Table 3.

All these effects except two represent *a greater amount of activity* in the function being measured. (It seems the vasoconstriction represented in finger volume and volume pulse may be counted as such.) The exceptions are the two rate variables, for breathing rate and heart rate are both slowed by the stimulus. This slowing may be compensatory action for the "pressor" effects of the other variables. The heart rate effect was so interpreted in our analysis of circulatory variables. For breathing the case is not so clear; the same mechanism or another might be operating. So far as our measurements show there is no time difference between the increase of breathing amplitude and the decrease in rate. Both are rather late in developing.

The sequence of events following the first stimuli seems to be:

1. Skeletal muscle tension increase (max. mean 0.2 to 0.3 seconds).
2. GSR (max. mean estimated 4 seconds).
3. Vasoconstriction (min. mean 5 to 10 seconds).
4. Interpulse interval increase (max. mean 5 to 10 seconds).
5. Pressure pulse increase (max. 5 to 10 seconds).
6. Breathing change (max. mean 15 to 20 seconds).

The systolic pressure increase is not exactly located in time, but it definitely precedes the maximum of the mean breathing changes. If latencies are considered rather than times for maximum means, the order is the same except that the slow-down in pulse rate is then

TABLE 3
Nature of Response to Initial Stimulus in Variables Recorded

Variable	Effect	Latency
Skin resistance	Decrease	2.01 sec. (mean reciprocal reconverted)
Pressure pulse	Increase	
Breathing amplitude	Increase	
Breathing cycle time	Increase	
Systolic pressure	Increase	
Finger volume	Decrease	8.7 sec. (mean reciprocal reconverted)
Interpulse interval	Increase	
Skeletal muscle tension	Increase	Approx. 0.1 sec.

found to begin 2.5 seconds later than the vasoconstriction effect, a fact which is in accord with the theoretical analysis.

There is a rough similarity in the order of the above list and the order of adaptation effects. The first four variables show significant adaptation decreases, the fifth shows a nonsignificant decrease, and the breathing response shows an increase, significant in the case of amplitude. That shorter latency phenomena show adaptation may be a general principle.

For all the variables the effect of the noise stimulus is in the same direction as that produced by the questions in a lie detection experiment (16). In two respects the response differs from the response to shock threat in that experiment. The threat of electric shock depressed the breathing amplitude and had little effect on rate. (As compared to truth-telling, lying likewise depressed the breathing-amplitude response.) The mean response to the noise stimulus is in general less than it is to lie-detection questions.

The main question of this experiment is whether the general autonomic and skeletal muscle response to the stimulus changes with stimulus repetition. That is, is the pattern of the various responses the same after a series of stimulus occurrences as it is after the first occurrence? For the pattern to remain constant, each of the variables in question must be affected by stimulus repetition in the same way to the same degree. If it had been the case that habituation or lack of it had been manifested by all variables under consideration, a rigorous specification of what is meant by "affected to the same degree" would have been necessary. In the present case, however, it is sufficient to point out that some of our variables (skin resistance, volume pulse, and interpulse interval) show a smaller response, some (pressure pulse, time of breathing cycle, systolic pressure, and finger volume) give little evidence of a change of response, and one (breathing amplitude) shows a larger response after repeated stimulation than after the initial stimulus. Under these circumstances, it is evident *that a change in response pattern has occurred.*

In view of the different behavior of the several variables, it is clear that no one of them could be taken as an index of the activity of the autonomic system, or even of one of its major divisions. The explanation of the differential repetition effects presents a physiological problem. It can hardly be attributed to exhaustion effects in the effector organs, since it is clear from the irregularities of some of the response curves that large responses can be made late in the series. This leaves the possibility of differential excitability (a rising threshold) either in the effector organs, in the particular efferents supplying them or both. It may be presumed that differential extinction cannot be referred to any part of the response systems which are common to the several variables. This would seem to eliminate the afferent limb of the response system from consideration.

It is a common impression that autonomic responses are more susceptible to extinction than skeletal muscle responses. Rapid extinction is thought to be one of the peculiar difficulties of GSR studies, for example. The comparison of data made in this study suggests that skeletal muscle responses, of a certain sort, have the most rapid extinction. It may be that other conditions, such as the presence or absence of some sort of reinforcement, have led to the opposite impression.

The present results on both latency and extinction make it clear that the

autonomic responses cannot "support" one skeletal muscle response of comparable type. On the basis of the response latencies one might suppose the reverse: that the autonomic responses are "supported" by the skeletal muscle response, but the relatively rapid extinction of the muscle response seems to rule out this possibility.

E. Summary

The effect of stimulus repetition on the responses of nine autonomically controlled variables and their combination was investigated experimentally and compared with the previous results on skeletal muscle response. Seventeen *Ss* were each given a series of ten identical auditory stimuli at one-minute intervals. In each case the relation of response to preresponse base level was studied. A comparison of extinction effects was made, with responses being expressed as percentage of maximum mean responses and with the responses being considered by a rank-order method (refer to paragraphs 4 and 5 under "Results").

It is concluded that there are differing effects of stimulus repetition for the several responses: GSR, interpulse interval, and volume pulse show an extinction effect; breathing amplitude shows an increasing response with repeated stimulation; and systolic pressure, pressure pulse, time of breathing cycle, and finger volume, as well as GSR latency and finger-volume response latency, show no effect. It is concluded that the pattern of response produced by autonomic action is changed by stimulus repetition.

IV. THE EFFECTS OF STIMULUS INTENSITY

It is easy to see, even in an informal demonstration of somatic effects, that the intensity of the stimulus is an important determiner of the responses. For certain of the responses (GSR and vasoconstriction, [26] and the muscle tension response [9]) this psychophysical relation has been studied, though for most the information is lacking. In the present experiment the object was to confirm and extend previous results on the point, and to determine the relation for some other variables so that any changes in the character of the response pattern can be made out. The experimental design will also permit an examination of whether responses to different stimulus intensities undergo the same adaptation changes as the stimulus is repeated.

The stimuli used were 1000-cycle tones of 120, 90, and 70 db. (referred to below as *A*, *B*, and *C*, respectively), delivered through earphones. (The decibel intensity measurement was made with an "artificial ear.") An automatic device presented a tone to *S* for two seconds at one-minute intervals. Each *S* was given each tone four times. *Ss* received the tones in various orders, but the orders were so chosen that for the whole group of 30 *Ss* each tone appeared in each serial position equally often, and for each *S* appeared once in each quarter of the sitting. (This procedure, of course, confounds the variance of order and the variance of *Ss*.) The *S* was told that he would be given some loud tones, and that he should remain as quiet as possible at all times.

The response variables recorded were:

1. Muscle action potential (MAP)—right extensor *digitorum communalis*
2. Muscle action potential (MAP)—left extensor *digitorum communalis*
3. Skin resistance from the left palm
4. Pressure pulse—left radial artery
5. Volume of tip of right index finger

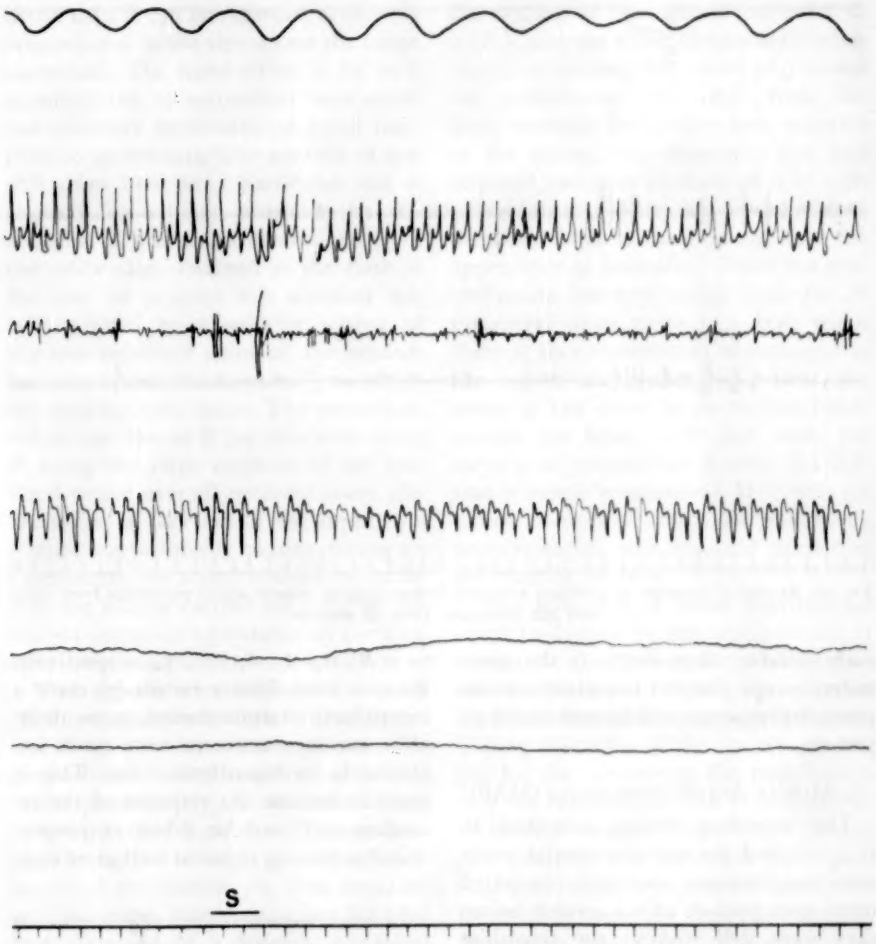

FIG. 4a. Record of response to auditory stimulus. In order from top, lines represent breathing, pressure pulse, ballistocardiogram, volume pulse, finger volume (constriction—up), chin volume (constriction—down), and time in seconds.

6. Volume pulse from right index finger
7. Chin volume
8. Chest breathing
9. Ballistocardiogram

A specimen set of records of these variables is shown in Fig. 4.

The blood pressure determinations used in the stimulus-repetition experiment were omitted from this one. Reanalysis of the preceding data showed, in addition to the effects reported, that the periodic application of occluding pressure seriously disturbed all the other variables of the circulatory system, and probably the blood pressure itself. It seems to confuse more information than it produces.

The results will first be presented for

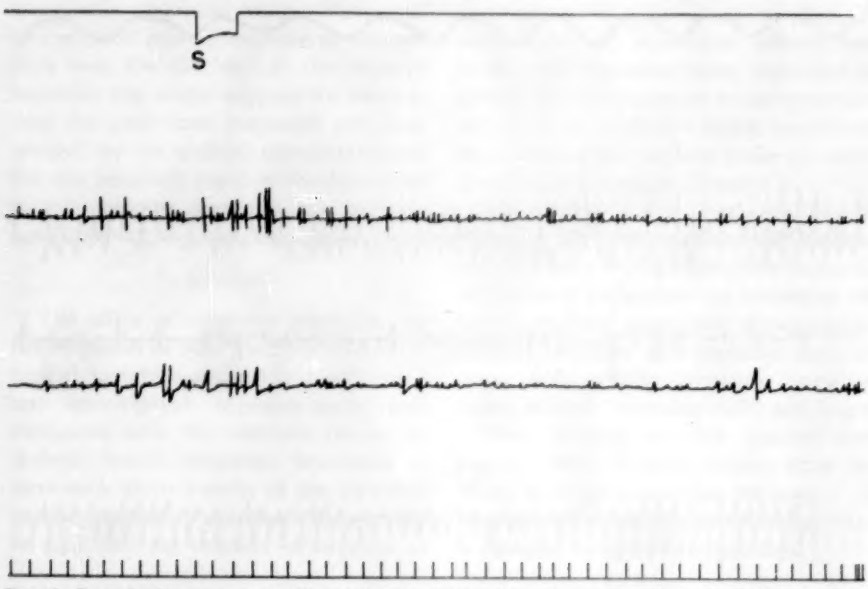

FIG. 4b. Record of response to auditory stimulus. Integrations of muscle action potentials from right and left forearms. Time in seconds.

each variable taken singly, in the above order, except that the two muscle action potential responses will be considered together.

A. MUSCLE ACTION POTENTIALS (MAP)

The recording device, described in (16), rectified the action potential waves after amplification and then integrated them over periods of 0.1 second before they were delivered to the recording styli. These, therefore, deflected each tenth of a second and the size of the deflection represents the sum of the muscle action potentials during the tenth of a second.

In the measurement procedure these deflections were sampled by reading the maximum deflection in each of the two one-second intervals before the onset of each stimulus and the maximum deflection in each of the following twelve one-second intervals. These will be referred to as B_1, B_2, A_1, A_2, . . ., A_{12}, respectively. Because some former results (7) made a logarithmic transformation seem desirable, our measurements were made immediately in logarithmic form. This is possible because the response of the recording styli can be taken as proportional to the log input as well as or even

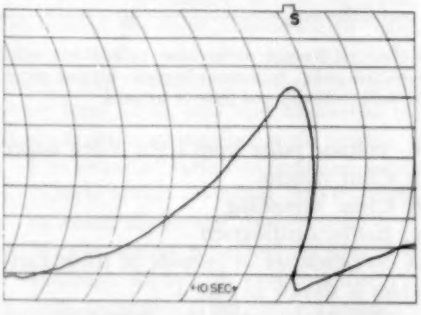

FIG. 4c. Record of response to auditory stimulus. Skin resistance—left palm. Read right to left. Time intervals, 10 sec. each.

better than it can be regarded as directly proportional to the signal over the range concerned. The hundredths of an inch in which the measurements were made can therefore be considered equal logarithmic increments. The amount of amplification used in a particular run is allowed for in this procedure by the addition of a constant determined from the calibration obtained at the time of the run. In practice this addition was accomplished by a suitable setting of the zero reference point on the measuring instrument ("Telereader") on which the readings were taken. The immediate values are thus in K log microvolt units, K being the slope constant of the line fitted to the over-all response curve (deflection as a function of log input). As K does not ordinarily change during an experiment, the actual multiplication by K is not usually carried out until all the desired statistical operations on the data have been completed.

Two reasons were adduced for using data in logarithmic form. There is, for one thing, a tendency for responses to be proportional to the base level of muscle action potentials (7). In the second place it seemed that logarithmic transformation brought a closer approach to normality of distribution (8). This latter result, of course, may or may not hold for data obtained in a different situation, and we therefore examined those from the present experiment with regard to distribution form. The values most in question are those which represent responses, i.e., change in level in action-potential level, and are obtained by subtracting a prestimulus reading from a poststimulus reading. In view of the duality of response reported earlier (7) *two such measures were used for each response*, one (*a*-response) being the first reading after stimulus onset (A_1) minus the reading of the interval just prior to it (B_2), and the other (*b*-response) being the third reading after onset (A_3) minus the prestimulus level (B_2). With the three stimulus intensities, four quarters of the sitting, two channels, and two response measures we have 48 cells each containing a distribution of individual measures. Most of these do not have the appearance of normality. There is a generalization, however, which holds for all instances: when there is a high mean there is an accumulation of measures at the top of the distribution; when the mean is low there is an accumulation toward the lower end; and when the mean is of intermediate size the distribution is nearly symmetrical. With this situation there is, of course, no monotonic transformation which would normalize the data in all cells.

The structure of these distributions could be formed by the superposition of a response distribution of variable mean but fixed standard deviation upon an invariant distribution of very flat form. Taking this view of the matter, an origin for the underlying flat distribution may be conjectured: the occurrence of accidental responses to stimuli coming just prior to the intended one. Such responses are indeed quite evident in the intervals between the controlled stimuli. No matter how careful the experimental control it would be too much to hope for their elimination. Unless these chance stimuli are in some way associated with the intended ones—and we believe we have avoided this experimental error—the mean differences, computed as we have done, would equal zero so far as they derive from these uncontrolled responses. This may well be the case in the distributions of our experiment. Standard deviations of the chance responses might be rather large since

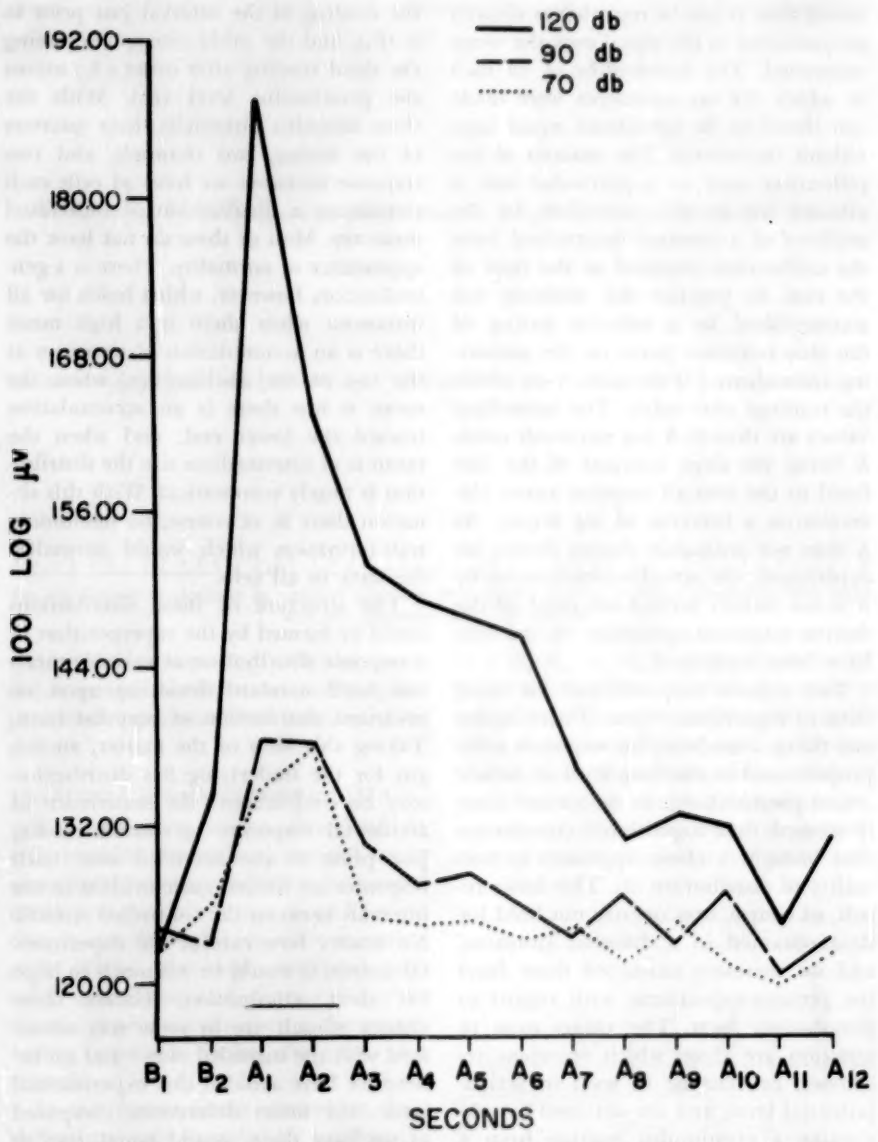

Fig. 5a. Mean muscle-action potential (right forearm) response to 1000-cycle tone of varying intensities. Horizontal line indicates stimulus duration.

the stimuli can vary in unpredictable fashion. This characteristic is quite evident in our data.

Averaging the readings for all *Ss* for each measurement before and after the three stimulus intensities gives the re-

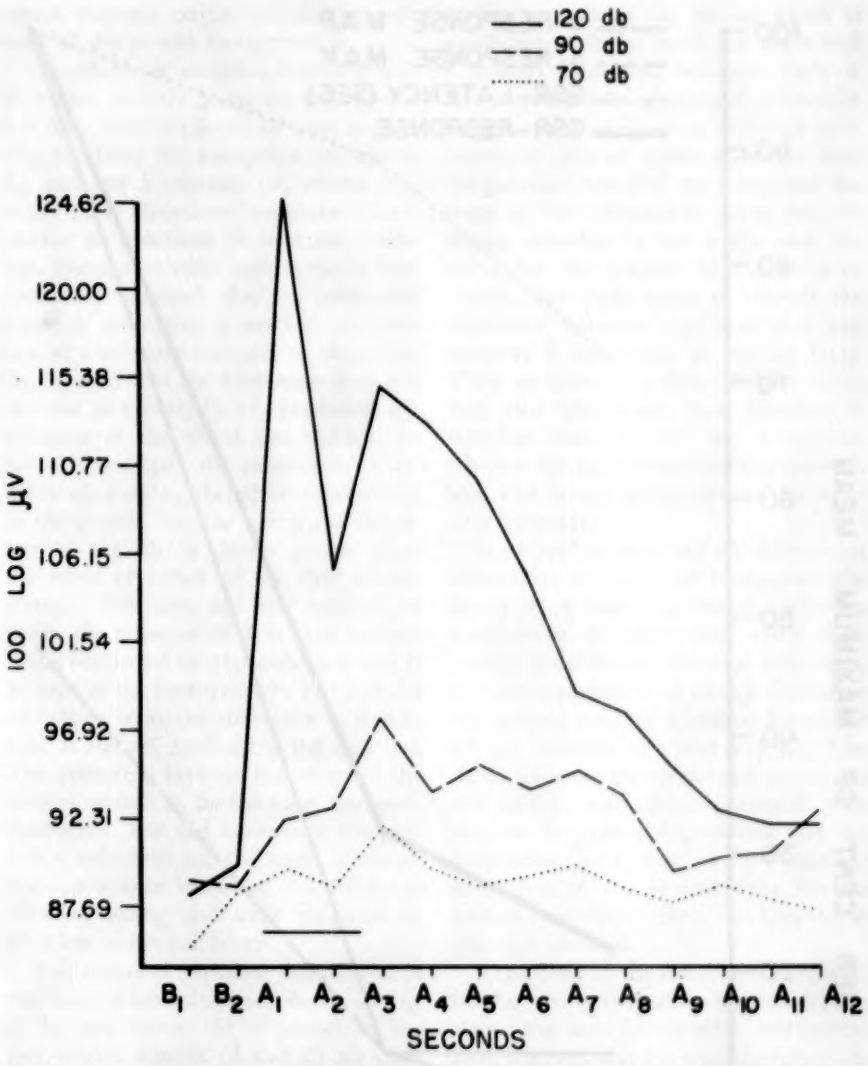

FIG. 5b. Mean muscle-action potential (left forearm) response to 1000-cycle tone of varying intensities. Horizontal line indicates stimulus duration.

sponse curves of Fig. 5a and 5b. These are very similar in form to curves previously reported (9). In the left arm following the strongest stimulus there is a definite second peak, which would correspond to the *b*-response previously described. In the right arm this appears to come earlier, so as merely to produce in the largest response at least a plateau (visibly starting at about A_3) in the decay of the *a*-response. The form of the recorded curve of muscle potential, of

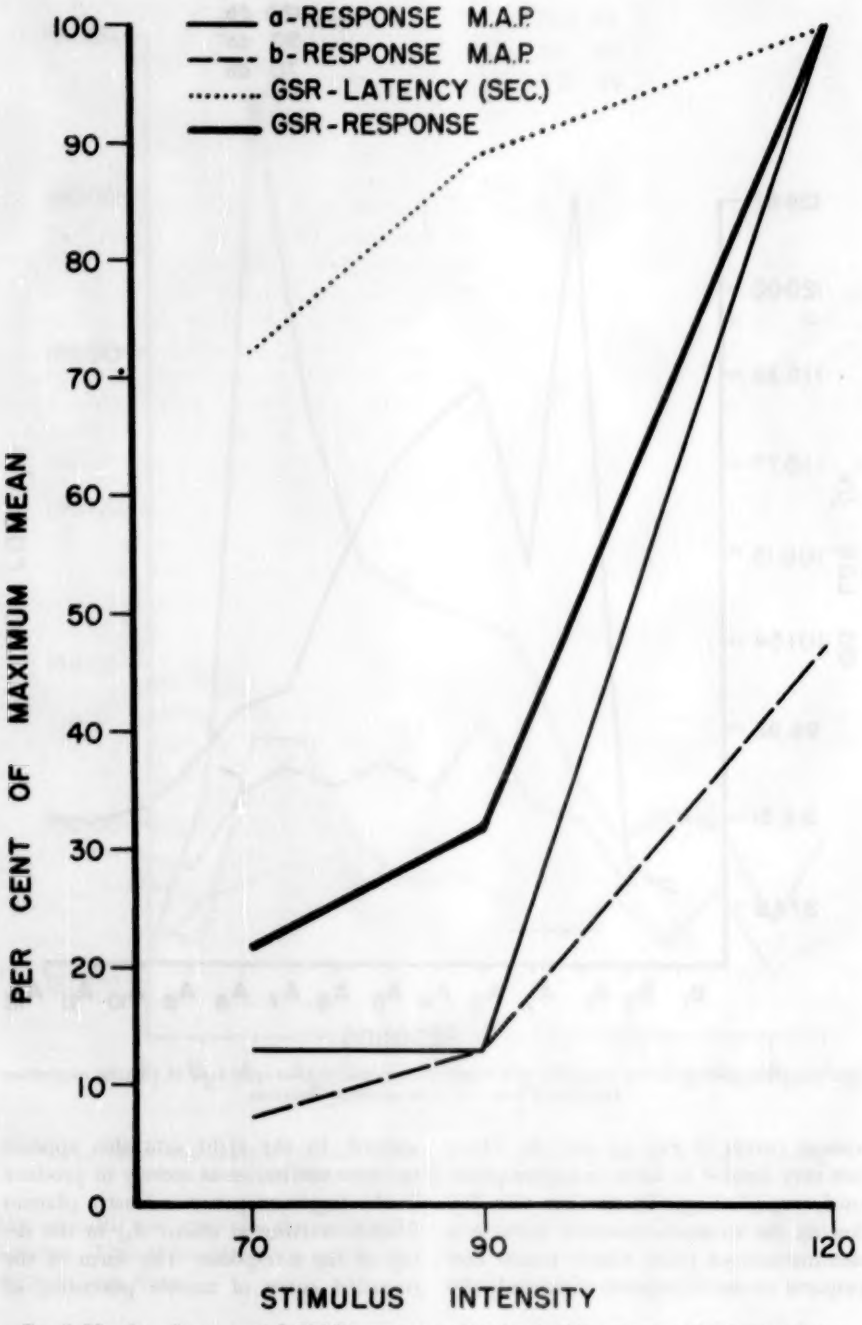

FIG. 6. Muscle-action potential (MAP) responses and galvanic skin responses (GSR) plotted as a function of intensity of a 1000-cycle tone.

course, depends on the relative proportions of the *a*- and *b*-responses.

The effects of stimulus intensity are, of course, exactly given in this figure. But they may be clearer in cross section (Fig. 6). Here the *a*-response (A_1 minus B_2) and the *b*-response (A_3 minus B_2), with both locations combined, are plotted as functions of stimulus intensity. The curves here confirm two effects previously reported: that the *a*-response function undergoes a marked acceleration at a stimulus intensity of about 100 db. (9), and that the *b*-response does not increase so rapidly (7, 11). Statistical significance of the effects was checked by the sign test (49): the proportion of individuals showing the effects represented in the graphs. For the *a*-response the effect of 120 db. is clearly greater than the effect of either of the two weaker stimuli. The sign test was applied 16 times, the response to *A* in each quarter being compared to responses to *B* and *C* in each of the four quarters. For 9 of the 16 comparisons the difference is significant at the 1% level using the sign test. The difference between the effects of the weaker stimuli is, by the same test, nonsignificant. For the *b*-response the general conclusions are the same, although the significance levels for the difference between strong and weak stimulus effects are somewhat lower.

The effects of repeated stimulation at the various intensities are shown in Fig. 7. In this figure the responses to the two weaker stimuli (*B* and *C*) are combined since there is so little difference between them. From the figure two facts about the *a*-response are apparent: for all stimuli together there is a pronounced adaptation, and most of the adaptation which occurs is an adaptation to the strong stimulus (*A*). For the stimulus *A*, the sign test shows a decrease from first to last quarter of the sitting which is significant at the .01 level. For the *B* and *C* stimuli combined, however, there is no demonstrably significant difference from first to last quarter, although each successive quarter shows a decline over the previous one. For the *b*-response the slope of the adaptation curve for the strong stimulus is less steep, and the curve for the weaker follows it more closely. For both types of stimuli the difference between the first and last quarters is significant at the .05 level. Thus we have the rather complex situation that adaptation is a function of stimulus intensity for the *a*-response, whereas for the *b*-response adaptation is less, and is not much affected by stimulus intensity.

In an earlier study (7) the differential adaptation of the *a*- and *b*-responses was observed. A later experiment yielded a comparison of adaptation rates (*a*-response) for different stimulus intensities. In that experiment, of design similar to the present one, the stimulus intensities all lay between 100 and 117 db. The mean response during the first quarter of the sitting was almost identical with that of the present experiment and the adaptation rate was nearly identical with that of the present data but no stimulus intensity effect on adaptation rate was detected.

Regardless of the difference in stimuli in the two experiments the adaptation rate is the same (*a*-response). Adaptation rate, it seems, may be conditioned on the mean size of the response, which happens to be the same in the two experiments. Two rules about adaptation are suggested by these experiments:

1. The size of the response elicited depends upon the number and size of previous responses.

2. Large responses are more affected

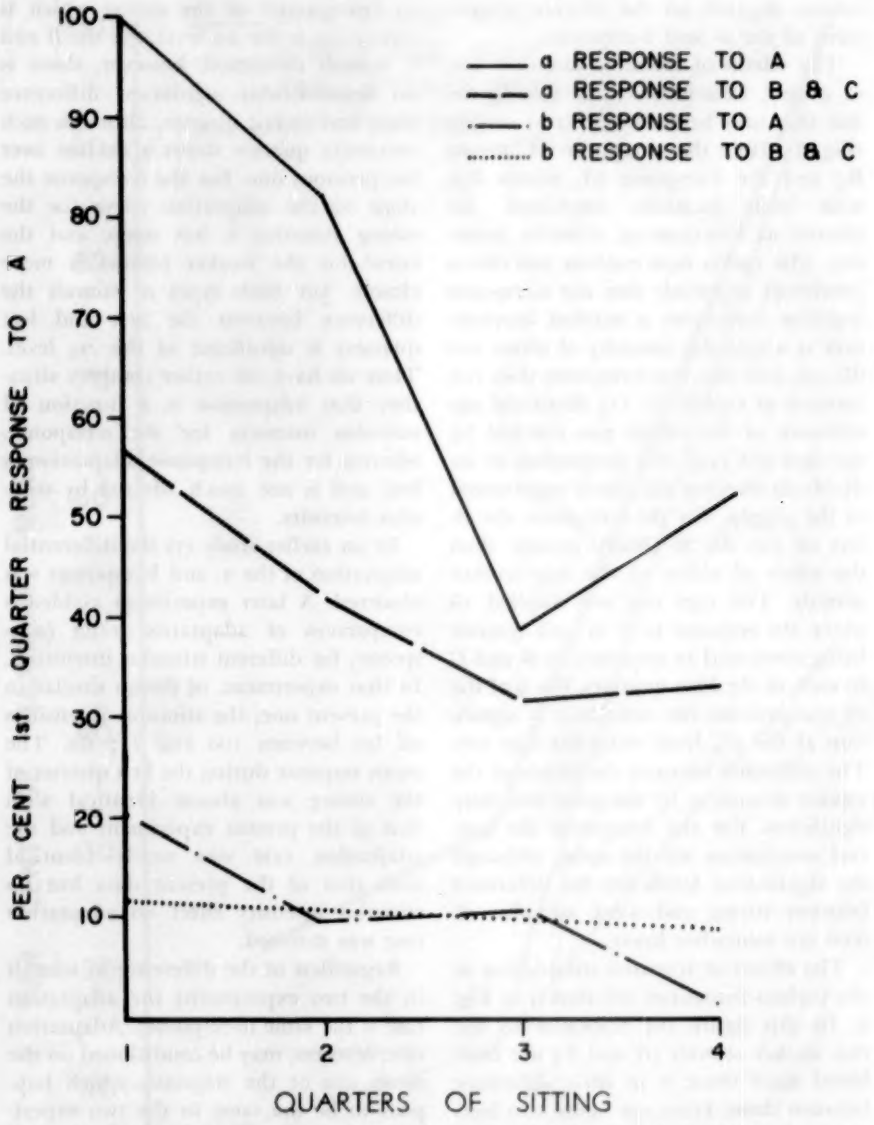

FIG. 7. Adaptation of muscle-action potential responses (*a* and *b*) to various intensities of a 1000-cycle tone presented in mixed order during a sitting. $A = 120$ db., $B = 90$ db., $C = 70$ db.

than small responses, so far as the *a*-response is concerned.

The first statement raises the question of whether adaptation is a linear function of the total of previous responses, or in other words whether we have an

TABLE 4
MEAN MAGNITUDE (PER CENT) OF GSR FOR VARIOUS INTENSITIES OF A 1000-CYCLE TONE

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	37.4	24.7	24.3	24.1	27.6
90 db. (*B*)	14.8	9.3	8.0	3.2	8.8
70 db. (*C*)	9.3	3.7	4.7	6.7	6.1
Mean	20.5	12.6	12.3	11.3	—

exponential decay curve in adaptation. Accordingly plots were made of response as a function of previous total response. There is a reasonably good approximation to a straight-line function in the data for *a*-response for all stimuli combined in both this and the former experiment, for the strong and weaker stimuli taken separately in the present experiment, and for the combined *b*-responses in the present experiment. These facts give support to the first generalization.

B. Skin Resistance

The Galvanic Skin Response Magnitude (GSR)

As in the repetition study, the GSR measure used is the ratio between (*a*) the minimum resistance after the stimulus, minus the resistance level at stimulus onset (base level), and (*b*) the base level, with the restriction that a decline in resistance must begin within 10 seconds after stimulus onset to be counted as a response.

The mean GSR (as defined in the foregoing) for all *S*s and all stimulus occurrences is .276, .088, and .061 for the 120-db., 90-db. and 70-db. stimuli respectively (Fig. 6). The four response ratios (one for each occurrence of *A*, *B*, and *C*) for each intensity were summed separately for each *S*, and these sums were used to test for intensity effects. According to the sign test (49), the response to the 120-db. stimulus is significantly greater than that to the 90-db. and 70-db. stimuli ($p < .001$, for both), and the response to the 90-db. stimulus is greater than that for the 70-db. stimulus ($p < .05$).

To study changes in response magnitude within a particular intensity, differences were taken for each *S* between GSR magnitude to first and last presentation for each stimulus intensity separately. The results for the strong and medium intensities are in accord with the results of the stimulus-repetition study. According to the sign test, there is a significant decline between the first and last presentation for the 120-db. stimulus ($p < .01$), and for the 90-db. stimulus ($p < .05$). For the 70-db. stimulus the trends are in the same direction but do not reach the .05 level using either the sign test or Wilcoxon's "signed-ranks test" (49). Table 4 shows mean values of GSR magnitude for each presentation of each stimulus intensity.

GSR Latency

The latency of the GSR was measured from the beginning of the stimulus to the point where a change of slope clearly indicated the beginning of a response (decrease in resistance). If no response took place within 10 seconds, the

latency was recorded as infinity.

As in the foregoing experiment the latency measures were converted to reciprocals so that they might be averaged. When they are averaged by stimulus intensity, we obtain the results shown in Fig. 6. To test the significance of the differences shown a mean latency for each stimulus for each individual was computed, and the sign test (49) applied to these distributions. The median reciprocal of response time to the strongest stimulus is significantly greater than the time for the medium ($p < .001$) and greater than the response to the weakest ($p < .001$). The responses to the medium and weak stimuli do not differ at any satisfactory level of confidence. There is evidently a curvilinear intensity relation here, as elsewhere, though it is not so steep as most of the others. This apparent difference in steepness would disappear if we were to choose a certain minimum latency as the true zero from which actual latencies should be measured. Then the latencies to the weaker stimuli, in terms of reciprocals, would be smaller fractions of the latency to the strong stimuli and the latency-intensity function would be brought into line with the other intensity functions. Such a minimum latency seems highly probable.

To study changes of latency with repetition, mean first latencies and mean last latencies were computed both for all stimulus intensities together and for each intensity separately. Individual scores were computed so that sign tests could be carried out here too. A curious result turned up. Considering all intensities together there is a decrease in latency reciprocals ($p < .05$), but for the response to the strong intensity alone there is an *increase* in latency reciprocals ($p < .05$). Taken singly the responses to the other two intensities show decreased latency reciprocals, but not statistically significant decreases. On the face of it there is a strange interaction between the intensity and repetition variables.

On looking for an explanation of this interaction we noted that the *A* stimulus hardly ever failed to evoke a response, while the *B* and *C* stimuli failed about 20% of the time. Where there was no response the reciprocal of the latency was, of course, treated as zero. Suspecting that the presence of these zeros might be the explanation we were seeking, we computed a set of means without counting them. These are the ones presented in Table 5. While there seems to be no way of testing significance here since some of the measures are paired and some are not, the trends for repetition all run in the same direction: there is an increase. We conclude that repetition of stimulation will increase or decrease the mean latency, depending on how many zero responses there are. If there are few zeros (or, of course, a constant number throughout) the early responses will be large, have long latencies, while the later ones will be small and have short ones. On the average this will be

TABLE 5

MEAN RECIPROCAL LATENCY $\left(\frac{1}{\text{SECONDS}}\right)$ OF GSR FOR VARIOUS INTENSITIES OF A 1000-CYCLE TONE
(Excluding Zero Reciprocals)

	Quarter of Sitting				
Intensity	1st	2nd	3rd	4th	Mean
120 db. (*A*)	.801	.778	.792	.850	.805
90 db. (*B*)	.698	.828	.819	1.102	.862
70 db. (*C*)	.684	1.179	.842	.904	.903
Mean	.728	.928	.818	.952	—

true of weaker stimuli as well, if the increasing proportion of zeros elicited by these stimuli is excluded.

Table 5 also makes us re-examine our conclusion about the stimulus intensity effect, for the general stimulus means run in the direction opposite to those we first arrived at. Here too the zeros are critical: without them we have an association of long latencies with stronger stimuli and larger responses. Though this is congruent with the trend during adaptation it may come about through a differential selection of cases, or indeed, by accident.

That there should, under any circumstances, be an inverse relation between amplitude and latency of this reflex is very puzzling.

An explanation is suggested by the facts of electrotonus (24, 14, 8). When a direct current is passed through tissues the polarization of cell membranes, such as those in the skin, is modified. In a state of positive electrotonus apparent tissue resistance is increased, the stimulus threshold is raised and electrical responses when they do occur are larger. Presuming that shorter latency indicates lower threshold, we may have a condition of lowered positive electrotonus as our series progresses, making the responses smaller and latencies shorter. If so, we should find the apparent tissue resistance to be less, concomitantly with smaller responses and shorter latencies. We have accordingly computed the means of the tissue resistances just prior to the stimuli (Table 6). They do in fact behave as the hypothesis states: there is a decline through the sitting. Further it is evident that where there is no response the mean actually increases. Apparently the threshold has increased in these cases.

TABLE 6
MEAN PRESTIMULUS RESISTANCE (OHMS)

Group		Quarter of Sitting 1st	Quarter of Sitting 4th	*N*
All *Ss*	Mean	97,850	78,838	30
	SD	82,950	69,260	
Ss with no response on 2 of last 3 trials	Mean	126,765	133,882	8
	SD	98,950	93,960	

In sum, we may say that where resistance decreases, latency and threshold and response size decrease. There is therefore good reason to suspect that there is a change in membrane permeability which has the same effect as that produced by applying more or less electrotonic current.

We should not imply, however, that this change in membrane permeability is wholly responsible for the decrease in response size. In our preceding experiment response decreased while the resistance increased (probably because the series did not include such a strong stimulus as the present). Latency in that experiment increased somewhat even when the no-response cases are disregarded, just as the foregoing hypothesis would predict.

The observations made, of course, refer to local peripheral events, but the conditions found there may also be symptomatic of similar conditions elsewhere in the system. Neural tissues as well as dermal are subject to electrotonus and are thought to change their permeability in the same fashion under normally occurring electric gradients.

C. Chest Breathing

Amplitude

In making records of the amplitude and duration of the breathing cycle the pneumograph was placed around *S*'s chest so as to secure as nearly as possible a maximum amount of movement. The sensitivity of the d.c. amplifier was then adjusted so as to produce deflections of several millimeters during normal breathing.

For measurement the records were

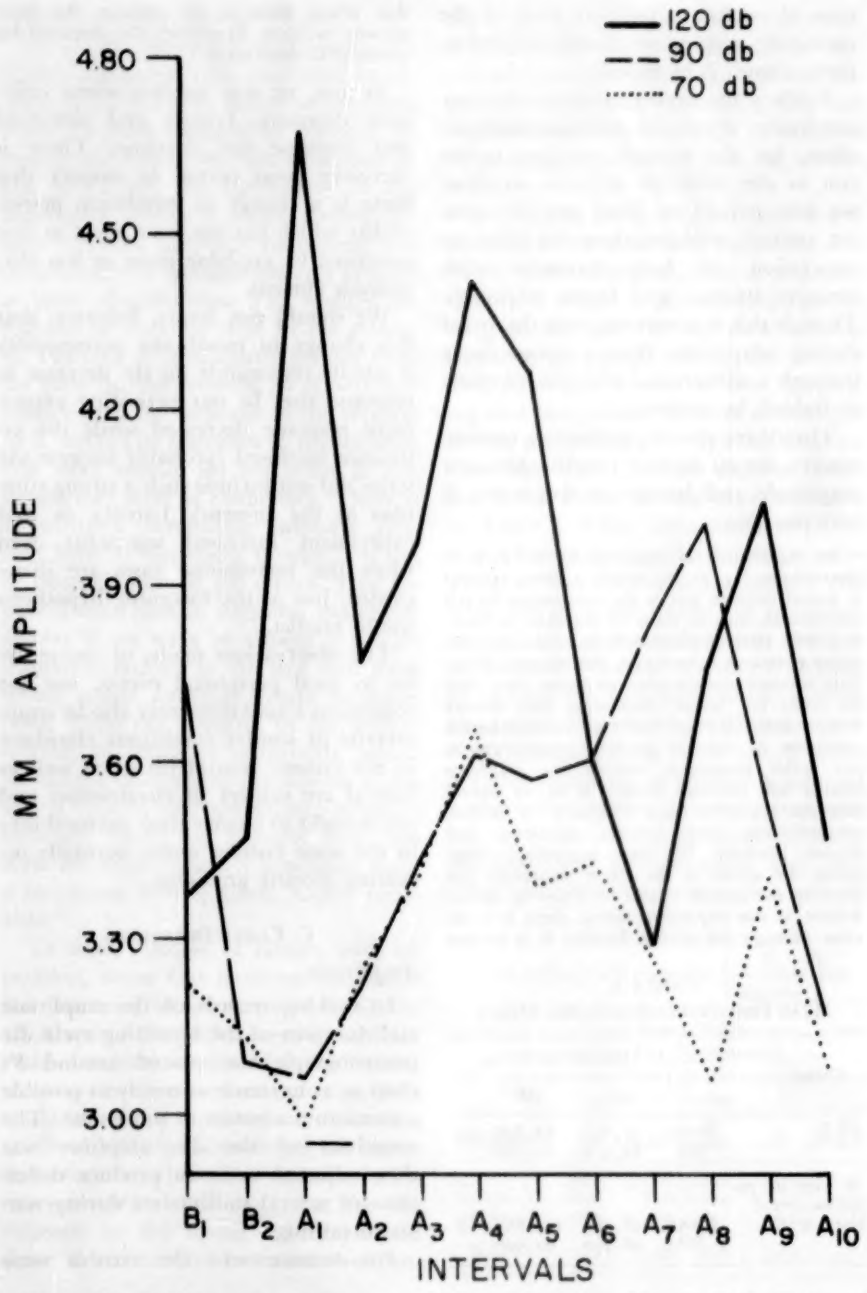

FIG. 8. The effect of a 1000-cycle tone of varying intensities on breathing amplitude. Horizontal line indicates stimulus duration. Intervals = 2.5 sec. each. Each point is the mean amplitude of 30 *Ss* to four presentations of a stimulus.

marked off into periods of 2.5 seconds, two before each stimulus and ten after, (referred to as B_1, B_2, A_1, A_2, . . . A_{10} respectively), and the maximum amplitude in each was measured. The means of these measurements for the four presentations of each of the three stimulus intensities are shown in Fig. 8. It is evident that the response to the auditory stimuli of the several intensities consists in an increase in amplitude of breathing, as it did in the foregoing experiment. The stimulus most like that used in the previous experiment was the *B* or 90-db. intensity. It may be seen in the figure that the response to it follows the same course that was found in the earlier experiment. The maximum is reached 15 to 20 seconds after the onset of the stimulus. But this is apparently not the case for the other stimulus intensities. The weakest evokes a response of similar latency but has an earlier maximum, while the strongest stimulus produces a maximum immediately. Some of the relationships change with repetition as will be shown below.

Because of the change with repetition it is necessary to study the response as a function of stimulus intensity at some one particular time. Fig. 9 shows this relation as it appears for the first presentation of each stimulus intensity for readings taken at the time used in the previous study: 15 to 20 seconds poststimulus ($A_7 + A_8$). Individual measures of response were secured by subtracting from these readings the prestimulus means ($B_1 + B_2$) for the cell block to which the reading belonged, i.e., the prestimulus mean ($B_1 + B_2$) for all *S*s for their first experience of the stimulus intensity in question. (So far as means are concerned the result would be the same as though individual prestimulus [$B_1 + B_2$] readings were subtracted in each case, but the effect is different when significance tests are applied. The reason for the procedure is that cell means of prestimulus measures do correlate with poststimulus measures, while individual prestimulus measures seem to have little correlation with corresponding poststimulus measures, as reported in the foregoing study.)

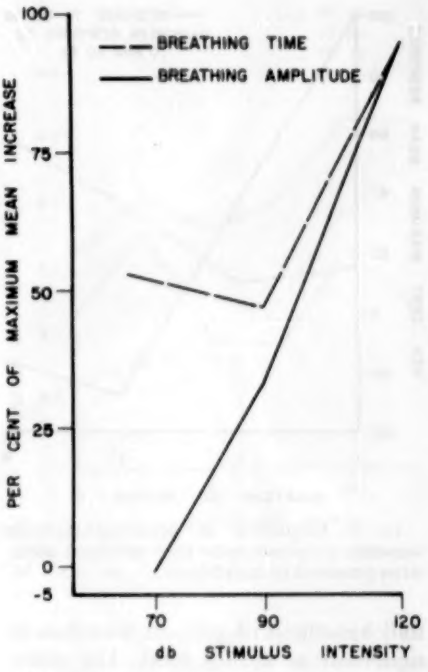

FIG. 9. Responses of breathing amplitude and duration of breathing cycle as functions of the intensity of a 1000-cycle tone. (Means of 30 *S*s to the *first* presentation of each intensity for amplitude. Means of four medians at each intensity for breathing time.)

Significance of the differences between the three means was tested by finding the percentage of individuals who gave a greater response in the one case than in another. When the response to *A* is compared with the response to *B* and with the response to *C*, the differences from a

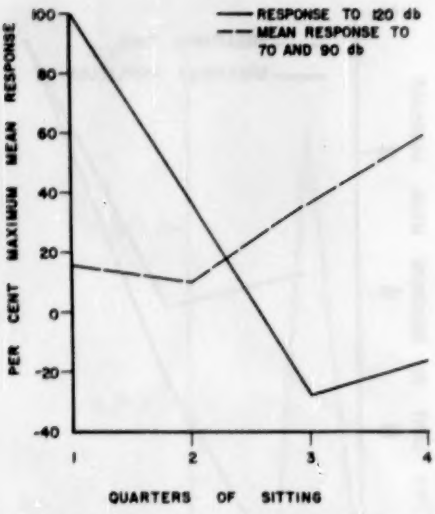

FIG. 10. Adaptation of breathing-amplitude responses to a 1000-cycle tone of several intensities presented in mixed order.

null hypothesis of 50% are found to be significant at the .01 level. The difference between the two weaker stimuli does not approach significance. Although the response for this variable appears to increase almost linearly with stimulus intensity it could well be that it really increases very little between 70 and 90 db., and very much between 90 and 120 db.

Change in the responses to the several stimuli was investigated by computing the mean response to the three stimuli for each fourth of the session in the manner already described. Responses to *B* and *C* were combined because they are similar in size and trend. The resulting means are shown in Fig. 10. It is surprising that the responses to stronger and weaker stimuli show opposite trends. The significance of these trends was tested by comparing the first and last readings. By the sign test (49) (binomial expansion, two-tailed test) the decrease for the strong stimulus is significant at the .01 level. By this test the increase for the weaker stimuli is not significant but the Wilcoxon signed-rank test, a more powerful, though nonparametric test (49), shows it to be significant at the .05 level. Similar trends are found when the response is measured right after the stimulus and at 7.5 to 10 seconds after it.

The latter finding, of course, is confirmation of the result reported in the repetition study (see p. 16), that the breathing-amplitude response, for a similar stimulus intensity, increases with repetition.

Duration of Breathing Cycle

For measurement of the breathing cycle duration, records were marked off into five-second intervals, one before each stimulus onset and five after it. (referred to as B_1, A_1, A_2, A_3, A_4, and A_5). Usually such an interval includes a complete breathing cycle, the duration of which was measured. If it did not, the measurement was made for the cycle which was most nearly complete, including the part of it that lay outside the designated interval. There were a few cases in which there was no breathing during an interval and readings for these were recorded as infinity.

This latter fact requires the use of medians rather than means for the combination of data and for some purposes a transformation of readings into reciprocals. Fig. 11 shows the trend in breathing cycle time (means of medians) for each of the three stimulus intensities. (There were four medians for each intensity, corresponding to the four occurrences of each intensity; each point in Fig. 11 is the mean of four such medians.) Effects for the three different intensities are very similar. The lengthening of the breathing cycle reaches a max-

imum 15 to 20 seconds after the stimulus onset, as it did in the previous experiment. To test the significance of this increase over the prestimulus (B_1) level the readings in question were converted to reciprocals, so that the four responses to each stimulus intensity for each *S* could be averaged. When this was done, the resulting scores for each stimulus intensity were compared with the mean prestimulus (B_1) reading for that intensity. (These three prestimulus (B_1) means differ very little). The sign test applied to the resulting differences shows the increase to be significant at the .01 level in two cases and at the .05 level in the other. This outcome is, of course, a confirmation of the fact reported in the "repetition" study.

All three of the graphs in Fig. 11 show an increase in breathing time in the five-second period right after the stimulus onset. This immediate effect is quite consistent for our *S*s, appearing in both the medians and mean reciprocals of nearly all of the 12 cells. But when the sign test (49) is applied the effect is found to be not statistically significant ($p > .10$). Evidently we should not expect to find this early response as a regular thing.

The response during the 15- to 20-second interval (A_3) is plotted as a function of stimulus intensity in Fig. 9. The size is computed by taking the difference between the prestimulus (B_1) medians and the 15- to 20-seconds median A_3) shown in Fig. 11. To test the significance of the differences the reciprocals were again used. As the difference between the two weaker stimuli (*B* and *C*) clearly amounts to nothing, the responses to these stimuli were averaged and this average change over the mean prestimulus (B_1) level was compared for each individual with the response to the strongest stimulus, *A*. By the sign test the difference between the response to it and the mean response to *B* and *C* is significant at the .02 level. We may say therefore that there is an effect of stimulus intensity above a certain level of intensity.

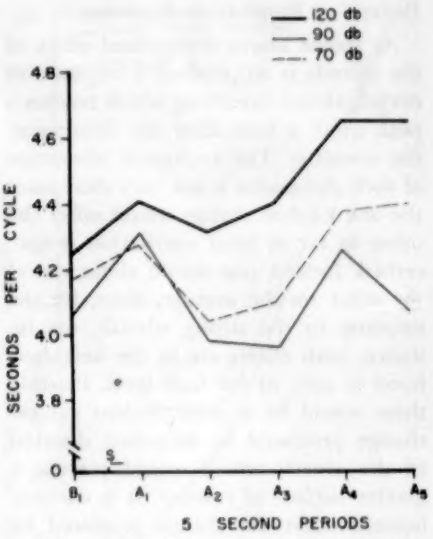

FIG. 11. Effect of a 1000-cycle tone on duration of breathing cycle. "S" indicates beginning of 2-sec. stimulus.

There are no consistent changes in this response produced by stimulus repetition, so far as our data go, for any of the stimulus intensities taken separately or for all taken together. Medians for the 12 cells are shown in Table 7.

TABLE 7
INCREASE IN DURATION OF BREATHING CYCLE FOLLOWING STIMULATION*

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	1.7	1.3	2.4	2.4	2.0
90 db. (*B*)	−.4	.7	2.3	1.2	1.0
70 db. (*C*)	1.5	2.8	1.2	.2	1.4
Mean	.9	1.6	2.0	1.3	—

* Median at 15 to 20 seconds poststimulus minus median prestimulus.

Pattern of Respiratory Response

As shown above the general effect of the stimuli is to produce a response of deeper, slower breathing which reaches a peak quite a time after the delivery of the stimulus. The biological advantage of such a response is not very clear since the one kind of change would offset the other so far as total ventilation is concerned. Indeed, one would about cancel the other on the average, since, for the response to the strong stimuli, for instance, both effects are in the neighborhood of 10% of the base level. Possibly there would be a more efficient gas exchange produced by increased dilation of the alveoli which would present a greater surface of contact. It is curious, however, that the change produced by the stimuli is in the direction of the slow deep breathing found in sleep.

Duration and amplitude changes are similar functions of stimulus intensity. In interaction effects they apparently differ, however, as do their latencies, and except for the weaker stimuli their time courses look different. The amplitude function varies more with conditions than does the duration function. One possibility for explaining the difference would be that the effects are also present in the breathing cycle duration but are obscured by errors of measurement. But against this notion is the fact that time measurements are usually less subject to error than are amplitude measurements. It seems probable, therefore, that there is a certain amount of independence in the two phenomena.

Duration of the cycle in ordinary breathing is believed to be determined by the Hering-Breuer reflex initiated by heightened intra-alveolar pressure (43). The afferent impulses for this reflex operate over a vagal pathway to inhibit the medullary center for inspiration, whereupon a passive expiration follows until the Hering-Breuer reflex ceases. If the vagal path is interrupted surgically a deeper, slower breathing results (the rhythm of which is thought to be set by a similar feedback from the pneumotaxic center). An interference with the vagal feedback could therefore produce the kind of breathing reaction we have described, but it would not account for the partial independence of the two aspects. It was previously reasoned (16) that decrease in feedback would produce slower, larger breathing cycles and that increased arousal of the inspiratory center would produce faster, larger breathing movements. Possibly both these mechanisms operate in our experiment, one partially offsetting the other so far as cycle duration is concerned.

D. Pressure Pulse

For the main purposes of this experiment the pressure-pulse record was divided into 2.5-second intervals, two before and eight after each stimulus occurrence (these are referred to as B_1, B_2, A_1, A_2, A_3, A_4, A_5, A_6, A_7, A_8, respectively). In each interval the measure taken was the vertical distance from the largest deflection upward to the largest deflection downward. This way of measuring turned out to be inferior, in the present case, to the method used on some of the same records for the analysis of circulatory variables. Trouble arose from a stimulus artifact in a portion of the records, an artifact which produced a shifting base line during the two measurement periods just following each stimulus. Readings for these two periods are inflated by the shifts and must therefore be left out of consideration. Fortunately, however, the response which we wish to consider lies in the later periods which are not affected by the artifact.

After being adjusted for any changes in the gain setting of the amplifier during an *S*'s run, the measurements were all expressed as percentages of *S*'s pulse size before any stimulus had been delivered.

Detailed analysis of the pressure-pulse response as a function of time has been given in an earlier section and need not be repeated here. The reading in the interval from 12.5 to 15 seconds (A_6), which contained the maximum readings, minus the mean of B_1 and B_2 readings, was taken as a measure of the response.

The relation between responses to the three intensities, expressed as usual in percentage of the greatest, is seen in Fig. 12. Responses to *A* are significantly greater than responses to *B* by the sign test ($p < .05$), but oddly enough neither of the other differences reaches the level of significance for the series taken as a whole. Table 8, showing the 12 cell means, discloses, however, a differential repetition effect. The difference between the first and last responses to the *A* stimulus is significant at the .01 level by the sign test, whereas there is no significant corresponding difference for either of the other two stimuli. (This fact corresponds to the lack of significant adaptation effect in the repetition study.) In view of this differential adaptation, the greatest difference between stimulus intensities would be expected on the first application of each. Accordingly we find that for the first application the response to *A* is significantly greater than the response to either of the other two stimuli.

Thus for the pressure-pulse response we find the usual curvilinear intensity relation, most evident on the first trial, and a significant adaptation effect for response to the strong stimulus. It is possible, of course, that there is a smaller adaptation effect for responses to the other stimuli which our experiment is not precise enough to establish.

TABLE 8
PRESSURE-PULSE RESPONSE (MEAN INCREASE) AS PER CENT MAXIMUM MEAN*

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	100	−8	17	27	34.0
90 db. (*B*)	−4	36	1	3	9.0
70 db. (*C*)	5	21	−20	1	1.8
Mean	33.7	16.3	−.7	10.3	—

* Decrease indicated by minus sign.

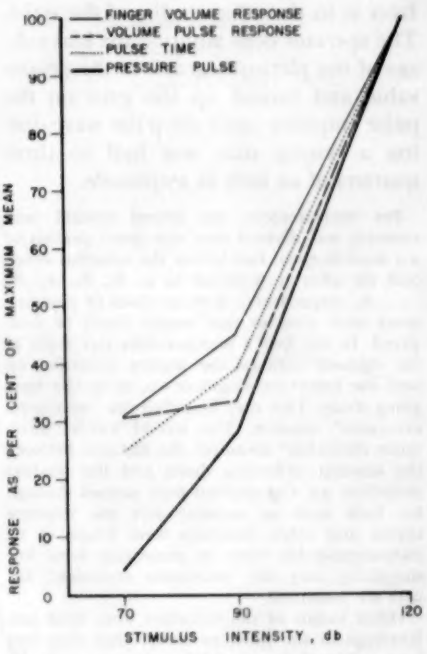

FIG. 12. Responses in several variables to a 1000-cycle tone as functions of stimulus intensity.

E. VOLUME PULSE

In recording the volume pulse from the right forefinger the electrodes were attached to the lower and upper sur-

faces as in the other studies of the series. The operator then adjusted the bias voltage of the plethysmograph to the proper value and turned up the gain on the pulse amplifier until the pulse wave during a resting state was half to three quarters of an inch in amplitude.

For measurement, the record around each stimulus was divided into our usual periods of 2.5 seconds each, two before the stimulus onset, and six after it (referred to as B_2, B_1, A_1, A_2, . . ., A_6, respectively). Two methods of measurement were used so that results could be compared. In the first a measurement was made of the distance between the highest excursion up and the lowest excursion down, as in the foregoing study. This may be called the "maximum excursion" measure. The second was a "minimum excursion" measure: the distance between the smallest deflection down and the smallest deflection up. Calculations were carried through for both sorts of measure and the response curves and other functions were found to be indistinguishable when in percentage form. For simplicity, only the "minimum excursion" figures are presented.

Other means of simplification were tried out. Readings in the previous study, after they had been adjusted for sensitivity changes made by the operator during a run, were converted to percentage of an *S*'s mean reading (for all stimuli) at 2.5 to 5 seconds prestimulus. In the present experiment the base for these percentages was the reading taken before the first stimulus only. Parallel calculations yielded the same results for the two methods.

Further, in the previous experiment the mean of readings 3 and 4 after each stimulus was taken as the basis for the response measure. Fig. 13 shows that in the present experiment the greatest response is in this same period, at reading 3 for the weaker and 4 for the stronger stimuli. Examination of individual sets of measurements showed that the greatest change occurred at 4 more often than at 3. The use of reading 4 alone rather than in combination was therefore tried out. Again a parallel calculation showed the same trends and same significances whichever basis was used.

Since Fig. 13 shows response curves much like those in Fig. 2, the time characteristics of this response seem pretty well established. The mean response has a latency of at least 2.5 seconds with some indication that there are longer latencies for weaker stimuli. For a strong auditory stimulus the decrease in size is about 20%, a value reached at 10 to 12.5 seconds after the stimulus onset. This point is followed by a slow recovery.

The intensity function was studied by finding the differences between the first means $\frac{1}{2}(B_1 + B_2)$ shown in Fig. 13 and the means at 10 to 12.5 seconds for the various stimuli (A_3). These are plotted as a function of stimulus intensity in Fig. 12, having been converted to percentage of maximum mean response. Again there is found to be a small increase in response size from 70 to 90 decibels and a much larger one from 90 to 120 decibels. The difference between the strongest and either of the two weaker stimuli is significant at the .01 level by the sign test, while the difference between the two weaker is not significant by either the sign test or the Wilcoxon signed-rank test. The increase in response from 90 to 120 decibels is more than proportionately greater than the increase from 70 to 90, and significantly so. There is evidently a nonlinear relation of response to intensity.

A decrease in response size with repetition of stimulus takes place in the present experiment, as would be expected from the results of the previous experiment. This effect, while showing in the column means of Table 9, is not significant by either the sign test, the Wil-

TABLE 9
MEAN DECREASE IN VOLUME PULSE FOR VARIOUS STIMULI
Per Cent Largest Mean Response

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	87	100	68	90	86
90 db. (*B*)	13	58	19	25	29
70 db. (*B*)	42	26	42	4	28
Mean	47	61	43	40	—

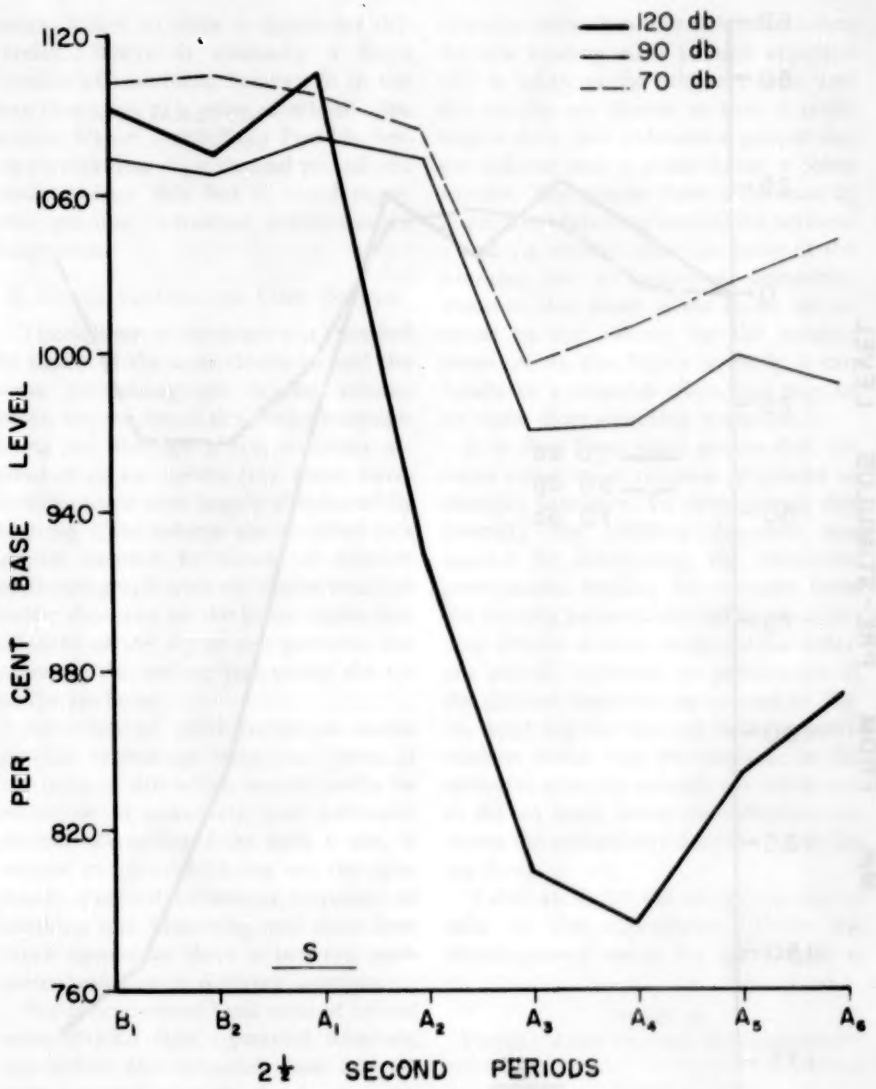

FIG. 13. Effect of 1000-cycle tone on volume pulse.

coxon test (49) or the *t* test comparing first and last quarter means. The experiment, of course, is not well designed for a precise test of this trend. No consistent interaction of repetition with stimulus intensity is apparent in the entries in the table.

In the previous experiment the repetition trend was brought out by the correlation of group means with serial position, whereas in that experiment as in this, comparisons of first and last

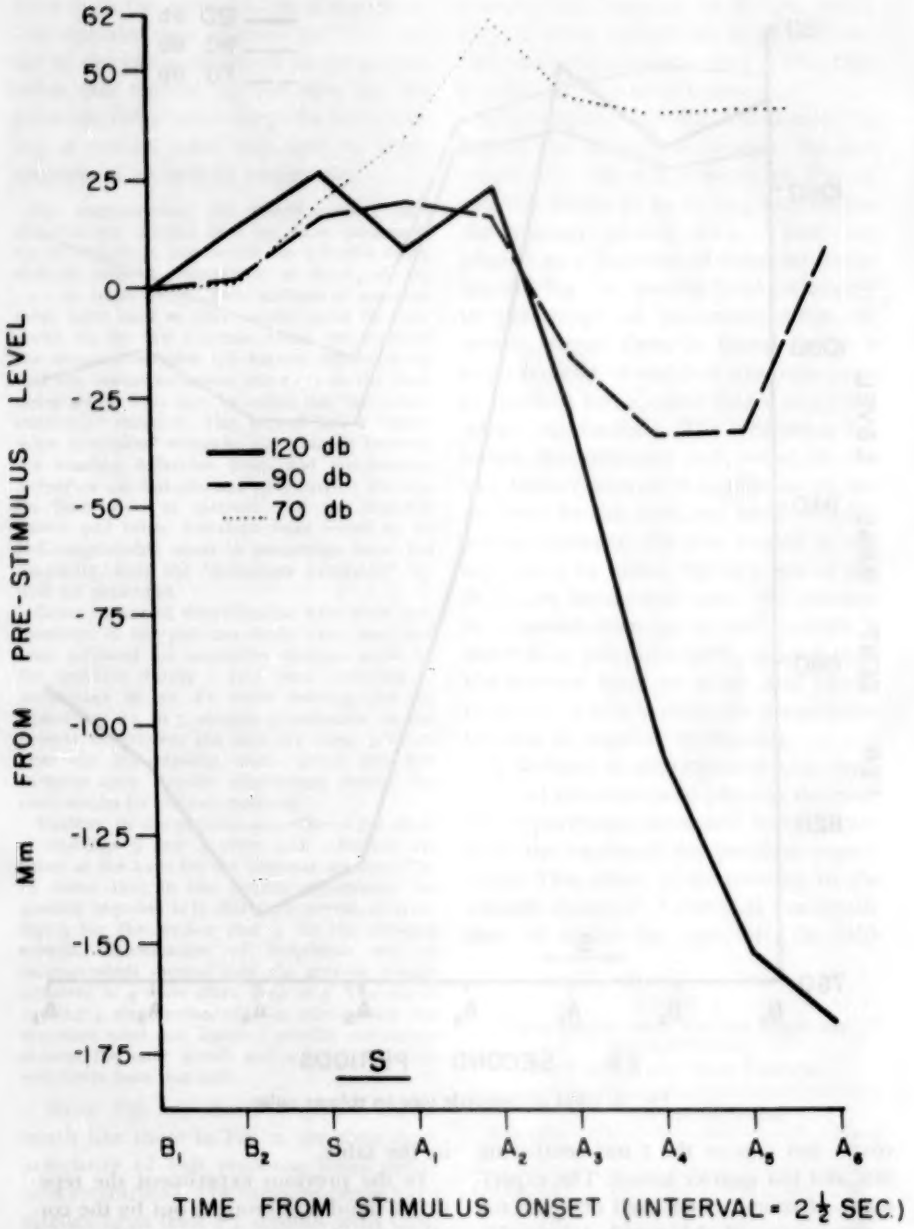

FIG. 14. Effect of 1000-cycle tone on finger volume. Downward change represents constriction. Two-sec. stimulus begins at S on base.

means failed to show a significant difference. There is evidently a large amount of variability between *Ss* in the way they react to a series of stimuli. (See section VI on variability.) Possibly better statistical or experimental procedures would reduce this, but it could represent genuine individual differences in adaptation.

F. Finger Volume and Chin Volume

The volume of the finger was recorded by means of the same electrodes and the same plethysmograph as the volume pulse, the output of the plethysmograph being fed through a d.c. amplifier instead of an a.c. system (10). Pulse waves in this record were largely eliminated by filtering. Chin volume was recorded in a similar manner by means of another plethysmograph with electrodes attached to the chin, one on the fleshy region just in front of the tip of the jawbone, the other on the surface just under the tip of the jawbone.

An electrical plethysmograph makes possible recordings from such parts of the body as this which would hardly be accessible to pneumatic and hydraulic devices. Recording from such a site, it seemed to us, might bring out the commonly observed autonomic responses of blushing and blanching and show how much agreement there is between vasomotor responses in different locations.

For measurement both sorts of record were divided into 2.5-second intervals, two before each stimulus onset and six after (referred to as B_1, B_2, A_1, A_2, . . . , A_6, respectively). At each division line a measurement was made of the height of the record line from an arbitrary base which was constant throughout all records.

Fig. 14 shows the mean of these measures for the finger volume for the three stimulus intensities. For this comparison the first reading made in each sequence (B_1) is taken as the arbitrary zero, and the graphs are drawn so that a point higher than this indicates a greater finger volume and a point lower a lesser volume. The graphs show a decrease in finger size beginning somewhere between 5 and 7.5 seconds after the onset of the stimulus for all stimulus intensities. Prior to this there seems to be an increase in the volume for the weakest stimulus. As this begins so early it can hardly be a stimulus effect, and may be no more than sampling variation.

It is clear from these graphs that the mean constriction response is related to stimulus intensity. To demonstrate this formally the intensity function was studied by subtracting the minimum poststimulus reading for response from the reading immediately before the stimulus. Means of such values for the different stimuli expressed as percentages of the greatest response are plotted in Fig. 12. Applying the sign test to these distributions shows that the response to the strongest stimulus exceeds the other two at the .01 level, while the difference between the two smaller does not reach the .05 level.

Table 10 shows the means for the 12 cells of the experiment. While the fourth-quarter mean for all stimuli is

TABLE 10
Finger Volume Response (Constriction)*

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	77	92	100	92	90
90 db. (*B*)	27	35	58	42	40
70 db. (*C*)	70	4	40	8	30
Mean	58	44	66	47	—

* Means of maximum decreases expressed as percentage of greatest mean.

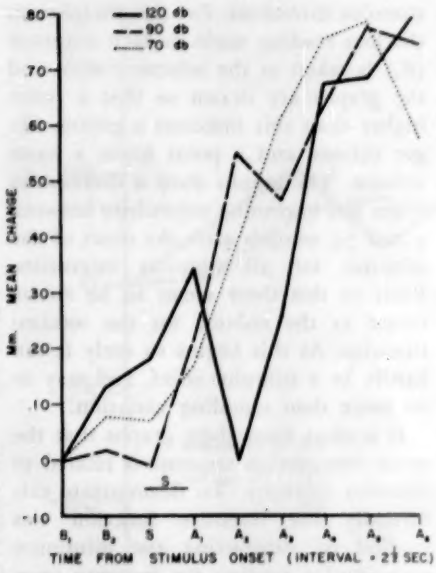

FIG. 15. Effect of 1000-cycle tone on chin volume. Upward change represents dilation.

less than the first-quarter the difference does not approach significance by the sign test. Nor are the trends found to be significant for the separate stimuli. In showing a nonsignificant over-all decrease the results are similar to those of the foregoing experiment.

All the results on the finger volume resemble a great deal those for the volume pulse in the present experiment. The two have been reported by Burton (4) to be similar functions, and such would seem to be the case in the present experiment except that the volume response is later than the volume-pulse response by about 2.5 seconds. This delay may be accounted for on the grounds that the vasculature becomes more resistant to pulse dilations before it actually decreases its volume.

The chin volume record sometimes shows a quick change in one direction or the other immediately after delivery of the stimulus, especially when the stimulus is of the highest intensity. These rapid changes can hardly be vasomotor; they are quite likely the result of muscular movements in the chin. For our purposes they are regarded as undesirable disturbances. Because of these it was necessary to discard the records of 6 of our 30 *Ss* for the *A* stimulus as being unreadable. The effect remains to contaminate the other measures, however, making for increased variability.

In this type of recording we must first ascertain whether an increase in the electrical impedance signifies a constriction or a dilation of the area. A test was made by rubbing the area vigorously to produce vasodilation and recording the effect. The direction turns out to be the same as for the finger: vasodilation produces increased resistance.

The means for this experiment show a consistent response trend (Fig. 15), which by its time characteristics seems to be vasomotor in origin. A sign test applied to the mean prestimulus and last poststimulus reading by *Ss* shows a significant difference at the .02 level. The direction of the mean response is quite opposite to that found in the finger area. For the typical *S* the response is vasodilation in the chin area, though there is a minority of *Ss* who consistently show constriction.

The curves in Fig. 15 show the response beginning within 2.5 seconds after the stimulus onset. This is a considerably shorter latency than evident in the finger volume response. The difference may be referable to a difference in the length of the efferent paths involved.

Fig. 15 shows plainly that at no single point of time after the stimulus is there any clear systematic relation between response size and stimulus intensity. When

TABLE 11
CHIN VOLUME RESPONSES (INCREASES)*

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	100	28	21	65	54
90 db. (*B*)	84	26	34	42	47
70 db. (*C*)	33	30	36	47	36
Mean	72	28	30	51	—

* Means of maximum changes expressed as percentage of greatest mean.

the data are treated by the alternative method used for the finger volume changes there is a systematic relation. In this method the reading at the beginning of the stimulus is subtracted from the maximum following the stimulus for each stimulus application. The means of these figures, for each of the 12 cells, are shown in Table 11. Although the column means for the three intensities are in the proper order neither the sign test nor the Wilcoxon signed-ranks test shows a difference between the categories at any acceptable significance level. Comparing the first quarter of the session with the last quarter does, however, reveal a decrease significant at the .05 level by the Wilcoxon test (49).

The vascular responses in the two regions studied are evidently very different. The chin reaction might be thought to occur passively under the force of increased blood pressure, as blood supply variations in the brain seem to occur. Although the vasculature in the brain is known to be innervated, passive vasodilation following constriction elsewhere is thought to be a common occurrence. Other cephalic vasculature could be similarly affected. Against this view, however, is the rather short latency of the chin dilation. The chin dilation seems to lead the finger constriction, rather than vice versa.

It seems more likely therefore that there is an active process of dilation of the tissues around the chin. Several mechanisms could be responsible for such a dilation: (*a*) decrease of tonic sympathetic activity, (*b*) activation of those sympathetic fibers which produce dilation, (*c*) activation of a parasympathetic channel so as to overcome whatever sympathetic activity may exist. The first is not excluded, but difficult to entertain, since it presumes two parts of the sympathetic doing opposite things.

The last alternative presumes a parasympathetic mechanism whose existence is doubtful (31). While one cannot yet decide just what the mechanism is, there is no doubt that it is different from that in the digits, and one cannot safely refer to over-all states of vascular constriction and dilation. Kuntz has pointed out that sympathetic constriction reflexes may be confined to part of the vasculature. The present results further show different parts of the vasculature affected oppositely.

Presumably there is in the whole body a net constriction effect, which would account for the rise in pressure pulse already discussed. Indeed the chin region might share in the constriction if the stimulus were still stronger. The beginning of this could account for the small (or nonexistent) difference in the effects of our several stimuli. It is, of course, well known that under some circumstances blood does leave the facial region.

G. INTERPULSE INTERVAL RESPONSES

The time between pulses was read from either the pressure-pulse or the volume-pulse record, whichever was the more convenient. The 2.5-second intervals marked off for the other purposes were also used for measuring the pulse

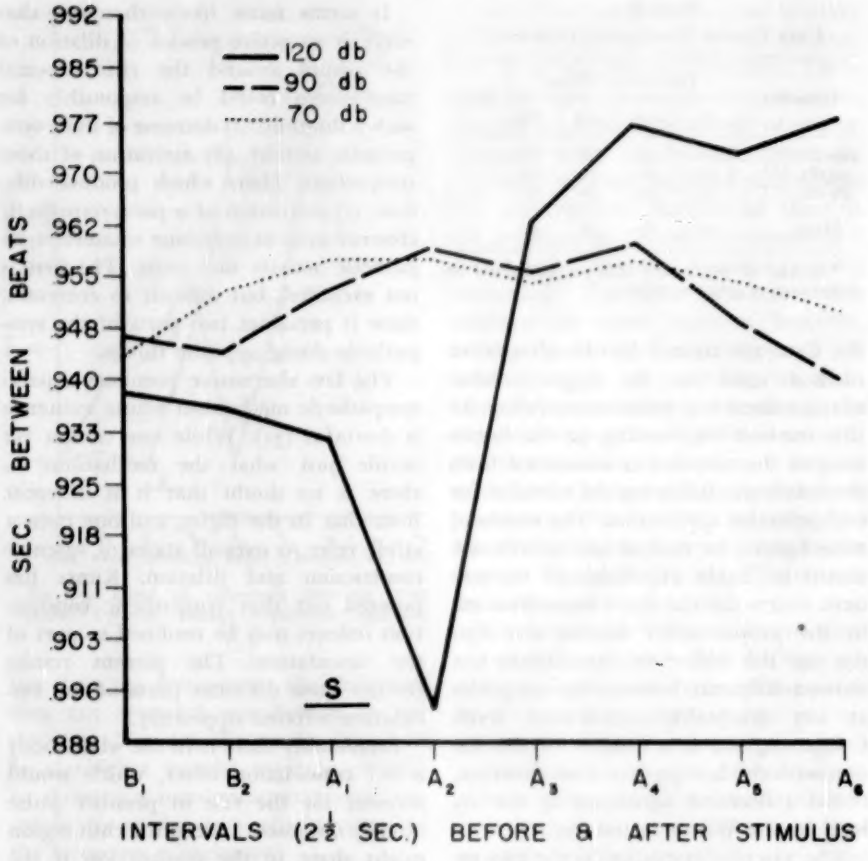

FIG. 16. Effect of 1000-cycle tone on interpulse interval.

time. In each of these the time covered by three successive pulse beats was measured, or in case there were less than three within an interval, the time measured was that covered by the three most nearly within the interval.

Fig 16 represents the means of these measurements. From these it appears that for all three stimuli there is a period of deceleration following the stimulus. This is the direction of the stimulus effect found in the repetition experiment and in a study by Dawson (12), as well as in the study of responses during deception. The maximum is reached rather late, 7.5 to 10 seconds after the beginning of the stimulus. In the response to the 120-db. stimulus there is evident an earlier phase of acceleration, a phase which was even clearer in the beat by beat analysis in the study of circulatory organization. Since it seemed possible that the method of measurement here used might be obscuring the acceleration phase of the response to the weaker stimuli, the beat-by-beat measurements used in

the study of the circulatory organization were re-examined so far as they were drawn from the data of the present experiment. The response curve for the two weaker stimuli, considered together, does indeed show a phase of acceleration which is greatest at the 5th poststimulus beat, as is the much larger acceleration for the strong stimulus. The maximum deceleration then follows at the 10th poststimulus beat, one beat later than it does following the strongest stimulus. The diphasic response pattern therefore seems to be characteristic of all three stimulus intensities.

Returning to Fig. 16 we see the maximum deceleration in the period 7.5 to 10 seconds after the stimulus onset (A_3). This period therefore was chosen as representative of the response. It is, however, 2.5 seconds later than the period used for the purpose in the repetition study. (In those data, however, the two periods 5 to 7.5 seconds and 7.5 to 10 seconds show almost the same mean values.) Taking the size of the response as given by the increase in time of beats from the prestimulus $\frac{1}{2}(B_1 + B_2)$ value to this period produces the stimulus-intensity-response function shown in Fig. 16 when the maximum response is taken as 100. The function is not greatly different from that of the other variables. By the sign test the response to the *A* stimulus (120 db.) is greater than the response to the *C* (70 db.) stimulus at the .01 level; the other two differences are not significant by this test.

Table 12 shows the mean responses for the 12 cells. Although the general mean response decreases with repetition, as it did in the repetition study, the decrease in the present case is not demonstrably significant. (It does, of course, tend to confirm the results of the prior study.)

The means suggest a difference in adaptation effects for the strong and the two weaker stimuli, but these trends do not approach significance when evaluated by the sign test.

TABLE 12
INTERPULSE INTERVAL RESPONSE

Intensity	Quarter of Sitting				Mean
	1st	2nd	3rd	4th	
120 db. (*A*)	51	45	100	80	69
90 db. (*B*)	54	30	18	7	27
70 db. (*C*)	76	14	−38	15	17
Mean	60	30	27	34	—

* Increases in interbeat interval in percentage of maximum mean. 100% = 165-ms increase.

H. THE BALLISTOCARDIOGRAM

"Ballistocardiogram" is a term which has been introduced to designate a record of the movement of the body produced as a recoil from the heart pulsation and associated phenomena (3). It is usually recorded from a subject lying on a special carriage, the movement of which is recorded by a mechanical system, which needs to be rather carefully engineered. The size of the deflection in the ballistic record is evidently related in some fashion to the force of the heart beat or the amount of blood ejected (stroke volume) although it has not been possible to determine just what the relation is. The possibility of responses in this variable seemed worth investigating. One would expect it to be rather closely related to our record of pressure pulse, though the factors affecting it are not necessarily the same.

We explored the possibility of recording this variable by means of placing a weighted crystal pickup on *S*'s wrist bone. Some sort of pulse effect can be detected by this means from a number of points on the skeleton, and the wave form does show the elements expected in the ballistocardiogram. Other elements, probably acoustic, are often present. Although individual *S*s show changes in this variable following stimulation, too few of the records are clear enough to make analysis possible.

SUMMARY AND DISCUSSION

The principal results of the experiment on stimulus intensity effects are detailed in Table 13.

In broader summary we may put the

TABLE 13
SUMMARY OF RESULTS

Variable	Response	Time (after stimulus) of maximum response	Stimulus intensity effect	Repetition effect	Differential effect of repetition
MAP (*a*-response)	Increase	1 sec.	Significant	Decreases	Sig. decrease for *A*. Nonsig. for (*B* & *C*)
(*b*-response)	Increase	3 sec. (?)	Significant	Decreases	—
GSR	Decrease in resistance	(Latency = 1.3 sec.)	Significant	Decreases	—
GSR latency			Significant*	Increase*	Sig. decrease for *A*.* Sig. increase for *B* & *C**
Breathing amplitude	Increase	15–20 sec. for weaker stimuli	Significant for 1st presentation		Sig. decrease for *A*. Sig. increase for (*B* & *C*)
Breathing cycle time	Increase	15–20 sec.	Significant	Increase (Nonsig.)	—
Pressure pulse	Decrease Increase	12.5–15 sec.	Significant		Sig. decrease for *A*. Not for *B* or *C*.
Volume pulse	Decrease	5–10 sec.	Significant	Decrease (Nonsig.)	—
Finger vol.	Decrease	12.5–15 sec.	Significant	Decrease (Nonsig.)	—
Chin vol.	Increase	12.5 sec.	Not significant	Decrease	—
Pulse time	Decrease Increase	2.5–5 sec. 7.5–10 sec.	Significant	Decrease (Nonsig.)	—

* Including "infinite" latencies.

results as follows:

1. The auditory stimuli, 70 to 120 db., produce a pattern of somatic response which includes all the variables measured. The response appears first in the skeletal muscles, where it quickly reaches its peak of increased activity and decays in a few seconds. Next in time comes the decrease in skin resistance, which has a wave form almost the same as that for mean log action potential for muscle, but about ten times the latency and much slower rate of change. A variety of circulatory responses is next in order: first an acceleration of pulse rate and decrease of pressure pulse, then a constriction of the finger vasculature and dilation of the chin vasculature, followed or accompanied by a slowing of the pulse and increase of force in the pressure pulse. Finally in the series comes the change to slower, deeper breathing. With the strongest stimulus, however, the deep breathing appears earlier in the sequence. For the particular pattern and sequence of somatic events produced by a 90-db. stimulus we should like to use the term *N-responses* as shorthand for the above list of changes. It is a response which can be regularly produced in the average *S* by using the procedure of the present experiment.

2. Each of the responses, with the possible exception of the chin volume increase, is affected in about the same way by variation in stimulus intensity. In terms of percentage of maximum response the reactions increase in a positively accelerated fashion with stimulus intensity over this region. The amount of increase with stimulus intensity is roughly the same for most variables.

The upward curvature of the intensity functions suggests that the response might be more simply related to the sound pressures than to the decibel values. We therefore plotted the functions, excluding GSR latency and chin volume, so as to show the relation log response = k (db.). (A plot linear in both directions would not distribute the three points very well.) On this sort of plot four of the nine functions are indistinguishable from straight lines. Of the remainder, two are inflected one way and three the other. It is possible that departures from linearity are but chance, although we have no exact test of them. At any rate the means of the percentage values, plotted in this logarithmic fashion, produce a line which is straight, departures from it being one or two percent. Since the log response (in these terms) is a linear function of decibel values we may infer that the response is a linear function of the acoustic pressure. This result is the same as Hovland and Riessen (26) obtained for GSR and finger volume constriction.

It is interesting that subjective loudness in sones over this region of the intensity scale follows a very similar function (46). A linear plot could be constructed by making somatic responses and subjective loudness the abscissa ordinate of a graph. Inasmuch as "subjective loudness" is a transformation of another kind of motor response it seems more reasonable to consider them parallel functions plotted on the same abscissa of stimulus intensity.

The original question of whether the response pattern varies with stimulus intensity can be considered on the basis of the intensity functions. If these are the same for all variables, we can conclude that the pattern of response remains the same while the response increases in size. In the results for stimulus repetition we found a definite difference in the function for the different variables. Here there is no clear difference in the slope of the intensity functions. Data with greater precision would be necessary to differentiate them. Possibly the chin dilation is an exception; in it intensity effects are small and not significant. There is also some indication that the intensity function for the muscle action potential a-response is steeper. With these reservations, that chin dilation may play a lesser role and muscular a-response a greater one, we would conclude that the pattern of response for the stronger intensities is much like that for the weaker: it differs principally in magnitude.

The effect of repetition of the stimuli is not so simple. In general, the present results confirm the conclusions reached in the preceding study. Changes are in the same direction here as in that study. For certain variables, breathing-cycle time change and finger volume response, showing a significant trend in neither study taken singly, the two experiments considered together make the trends more probable.

In our terms we may compare the amount of adaptation shown by the several variables in the two experiments. We have expressed this amount for each variable as percentage decline from first to last quarter in this experi-

ment and from first to last presentation in the simple repetition study. The question is, then, "Do the several variables show similar percentages of adaptation in the two experiments?" Seven variables are common to the two experiments, exclusive of GSR latency which probably should not be included in the comparison. For one of these, breathing amplitude, only the responses to the weaker two stimuli in the present experiment were considered, because of the interaction effect established for this variable. In these terms the mean amounts of adaptation found in the seven variables in the one experiment show a correlation of .86 (rho) with the amounts of adaptation in these variables in the other experiment. Evidently the various responses behave very consistently in adaptation even when the experimental conditions are rather different.

3. For certain variables, at least, the adaptation is specific to stimulus intensity also. For GSR latency there is a suggested explanation for the opposite trends encountered for higher and lower stimulus intensities. The opposite trends for breathing-amplitude response to the different stimuli is harder to account for but seem to be associated with different temporal patterns of response. There is evidently an interaction for the muscle-action potential response also, and there may be one for the pressure-pulse response, although the contrast in these two variables is between significant and nonsignificant trends rather than between opposite significant trends. In such instances as these the trends are converging: as responsiveness decreases, so do differences in stimulus effects. Trends of this sort would, of course, be necessary if adaptation for all stimuli is ultimately to bring the responses to zero at the same point. While responses do not necessarily arrive at a common zero, it might be a matter for remark that they do not do so more often.

The trends for breathing-amplitude response are divergent and some changes in their later course would have to be expected. As the lines cross in the middle of our sitting we find a strange condition at the end of the experiment, with the weaker stimuli producing a considerably greater response than the stronger.

In brief, finally, increased stimulus intensity over the range tested is found to increase the response in all variables (except one) about in proportion to the sound pressure; the differential effect of repetition on the several variables is confirmed, and for certain variables the state of adaptation is found to be specific to a stimulus intensity.

V. THE EFFECTS OF TACTUAL STIMULI

Casual observations suggested that tactual stimulation might produce response patterns differing from those seen in the preceding studies. This possibility was especially suggested during the investigation of the effect of applying occluding pressure on the upper arm upon vasomotor tone in the extremities. It will be recalled that the typical effect was contralateral and ipsilateral vasodilation instead of the more characteristic vasoconstriction seen in the experiments with auditory stimulation and generally reported as the effect of painful or intense stimulation (18, 37, 38).

As mentioned earlier, the present study is concerned with qualitive rather than intensive stimulus variation. A

series of stimuli—heat, cold, pressure, prick, and tickle—were applied to small areas of the skin. A search of the literature indicated that there is reason to expect the vasomotor response patterns to some of these stimuli to differ from those in the preceding experiments and perhaps to differ among themselves. Comparative evaluation of the various findings, however, is rendered difficult by at least three factors. We are concerned here with short-duration stimuli, or inputs, and with rather immediate responses, while the majority of other studies, particularly in the medical literature, have investigated the more continuing aspects of vasomotor regulation. Finally, the area of skin stimulated usually greatly exceeded that in the present study.

Procedures most closely approximating the conditions of the present experiment have been reported by Nafe and Waggoner (37), who found, using a water plethysmograph on the hand, that if the opposite hand is placed in water the contralateral response is constriction if the water is cool, dilation if it is warm, and constriction if it is uncomfortably hot. These investigators reproduced the constriction response to hot water in a second study which also showed contralateral constriction in response to electric shock and to pricking of the skin with needles (38). Waggoner reported that heating or cooling the hand in water did not produce contralateral skin temperature changes (47). It is known, however, that this effect may be modified by body and ambient temperatures (33, 42, 50).

Heating of large areas of skin produces vasodilation in the extremities (29) but it is in dispute whether this effect is reflex or depends upon the flow of warmed blood to the temperature-regulating centers (17, 42).

The vasodilation indicated by the reddening usually observed in skin exposed to extreme cold is reproduceable in the laboratory (33, 50) and appears to be accompanied by reflex vasodilation elsewhere. Here again the mechanism is in dispute and seems to involve local axone reflexes (33) as well as central reflexes whose outflow is along autonomic pathways.

We may point out here that most attempts to investigate the effects of blood temperature changes have involved occluding the blood flow of a thermally stimulated limb, the rationale being that vasomotor activity in other parts of the body will then not be affected by impulses arising from the temperature-regulating centers due to the return of heated or cooled blood. As mentioned earlier, however, serious modification of vascular tone attends pressure application and thus this procedure does not permit direct evaluation of the remote effects of thermal stimulation.

A more direct attack on the physiological mechanisms has been reported by Lynn and Simeone (34), who found that, in the dog, distension of the femoral vessels does not yield a contralateral vascular response but that, if the contralateral autonomic pathways are intact, stimulation of the perivascular tissues is effective even subsequent to ipsilateral sympathectomy.

There is, then, no clear picture available of the effects of qualitative variation of tactual stimuli upon the immediate vascular responses. Further, there appear to be no suggestions of what effects we may expect to find in the other somatic responses measured in this series of experiments. In view of this situation, the experiment to be reported was essentially exploratory and the selection of stimuli and their intensities was dictated primarily by considerations of convenience and simplicity.

Method

Subjects: Twelve Indiana University students, all apparently in good health, served as *Ss*.

Apparatus: The *heat* stimulus was provided by a GE type 47 miniature lamp mounted in a wooden holder and applied with a vertical force of 25-30 g. Throughout each sitting the lamp was connected to a standard three-cell storage battery. The temperature of the glass envelope in air was 97 ± 5°F. An identical unit, not connected to the battery, was used as a *control* stimulus. Between applications the stimului were not allowed to contact other objects but remained in free air.

The *cold* stimulus was a 3¼" length of ¼" brass rod with one end ground to a blunt conical point. Between stimulations this rod and its attached plastic handle were maintained in a tray of water and melting ice. Fifteen seconds prior to each application the unit was wiped dry. Application force was 25-30 g., the rod being held vertically with the pointed end in contact with the skin.

The *pressure* stimulus was a disc 3 mm. in diameter applied with a spring sphygmometer using a vertical force of 775-825 g.

The *prick* stimulus was a standard (3 mil) steel phonograph needle attached to the spring sphygmometer and applied with a vertical force of 350-360 g.

The *tickle* stimulation was accomplished by brushing an ostrich plume lightly to and fro over the skin.

None of the stimuli produced electrical artifacts, but there were uncontrollable mechanical artifacts due to movements by *S* in response to application of stimuli. The latter, except in the ballistocardiogram, only infrequently interfered with the measurement of records.

Procedure: Each stimulus was applied twice to each *S*. The order of application may be determined from Table 14, where each column shows the order of application for two *S*s, one receiving twelve stimulations in the order indicated by reading down and then back up the column, the other having the stimulus order determined by reading up and then down the column. The *S*s were randomly assigned to stimulus orders.

TABLE 14
STIMULUS ORDER

A	B	C	D	E	F
B	C	D	E	F	A
C	D	E	F	A	B
D	E	F	A	B	C
E	F	A	B	C	D
F	A	B	C	D	E

A = heat, B = pressure, C = tickle, D = heat control, E = prick, F = cold. Two orders are represented by each column, one reading down and up, the second reading up and down.

Each stimulus was applied for five seconds to the white skin of the pronated left forearm at a point midway between the elbow and wrist. The interstimulus interval was one minute.

Pickup instruments were attached as in the previous experiments, with the exception that the arterial pressure pulse as well as the ballistocardiogram were taken at the right wrist. The GSR electrode clamp served as a cradle to stabilize the position of the left arm.

After the instruments had been attached, the *S* was assured that no electric shocks would be given and then *E* touched *S*'s left forearm at the appropriate location and told *S* that he was trying to find out the effects of several different kinds of skin stimuli when applied to this part of the arm. The *S* was then blindfolded and instructed to sit quietly for about ten minutes and then the experiment would begin. During this period, while the operator was adjusting the recording instruments, *E* sat quietly beside *S*. After the apparatus was adjusted, *E* applied the stimuli in order, with as little movement and sound as possible.

RESULTS

The response variables measured were the same as in the previous experiments with an important exception. Because of electrical shielding problems it was necessary for *E* to activate the stimulus marker by pulling a cord which operated a microswitch outside the shielded room. Although this technique appeared to give reasonably accurate indication of

stimulus application it was not considered sufficiently reliable to permit measurement of latencies. This problem was also responsible for the method of dividing records into intervals for measurement. The reference chosen was the middle of the stimulus mark and intervals were marked off about this point. The muscle-action potential records were divided in a way which will be explained later but the division of most of the other records may be conveniently detailed here. The records were divided into ten successive 2.5-second intervals, three immediately preceding the middle of the stimulus mark and seven subsequent to it. These intervals were numbered in order, B_1, B_2, B_3, A_1, A_2, . . . , A_7. These designations will be referred to later and the accompanying graphs will furnish convenient references to the position with respect to the stimulus application.

In preliminary work it was evident that the ordering effects could hardly be assessed. In consequence, as will be seen in detail later, the corresponding measures from the two applications of each stimulus were averaged for each *S*.

The general plan in reporting the results is that used above. The response variables will be considered separately and then a final analysis in terms of overall response patterns will be reported.

Preliminary investigation indicated extreme deviation from normality in the data. In view of this, two distribution-free tests were used for statistical purposes. These were the Friedman analysis-of-variance-by-ranks test (49, p. 432) where several treatments were to be compared and the Wilcoxon signed-ranks test (49, p. 438) for comparison of pairs of treatments and evaluation of changes in response curves.

Muscle Action Potentials

The amplified muscle action potentials were integrated over 0.1-second intervals and these integrated potentials were recorded continuously during each sitting.

The records were measured by reading the maximum pen deflection in each of the 1st, 2nd, 3rd, 5th, and 7th 1-second intervals proceeding backward from the middle of the stimulus mark, and in each of the 1st through 4th, 6th, 8th, and 10th 1-second intervals subsequent to the middle of the stimulus mark. After adjusting for amplifier sensitivity, the corresponding pair of readings from the two applications of each stimulus were averaged for each *S*. For each *S* the responses to the various stimuli were converted to percentages of a prestimulus level which was the mean of the averages for the 5th and 7th 1-second intervals prior to the middle of the stimulus marks for each stimulus.

The mean percentage in the stimulated arm for all *S*s is plotted in Fig. 17 for each of the measurement intervals. The response does not appear to exhibit the *a*- and *b*-response characteristic described by Davis (9) and seen in the experiments reported above. This, of course, may reflect the small sample used here and the method of converting measures to percentages. It will be noted that the maxima occur about 4 to 8 seconds after stimulus onset, which is somewhat later than that seen in the adaptation and intensity experiments. In most cases the maxima appear too early to represent effects due to stimulus termination. The response in the nonstimulated arm is similar to that in the stimulated arm but is much smaller and the maxima are somewhat earlier, occurring from 2 to 5 seconds after stimulus onset. This may

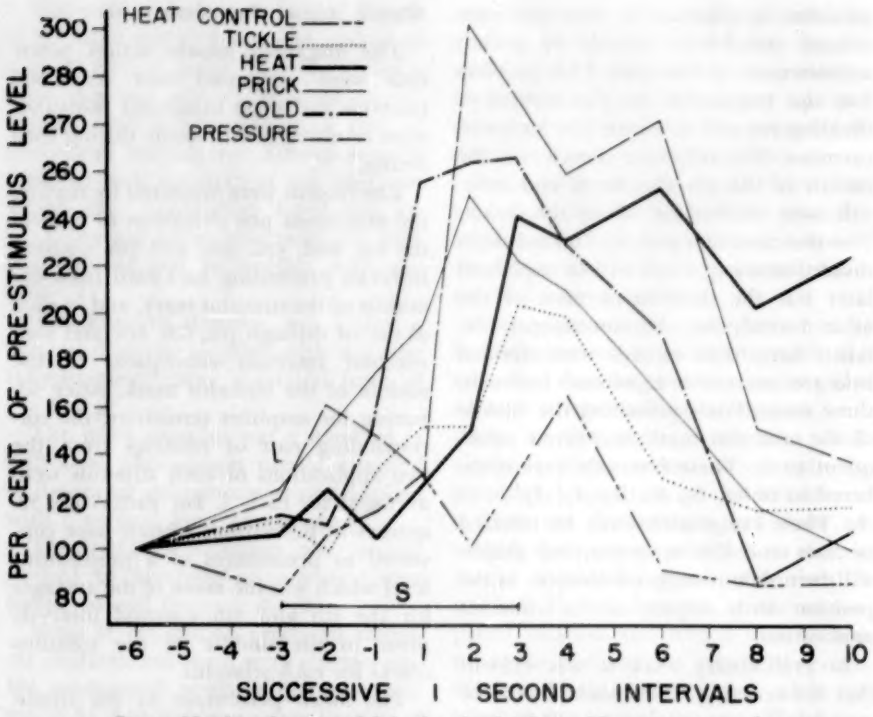

FIG. 17. Muscle action potentials in the stimulated arm. Mean percentages of prestimulus level are plotted for each stimulus.

possibly be taken to indicate that the response in the stimulated arm is complicated by tension adjustments made by *S* in meeting the force of application or, perhaps, in "shrinking" from the stimulation. In this way we could account for the unusually delayed maxima, but unfortunately the data are not adequate for analysis of this notion. We should, of course, have been surprised if the response to the relatively lengthy stimulation used here had turned out to have the same features as the response to short auditory stimuli.

Statistical assessment of the increases was made by subtracting 100% from the average of the percentages for the second and third 1-second intervals after the stimulus mid-point, and applying the Wilcoxon test to the set of percentage changes ($n = 12$) obtained for each stimulus. Other possibilities were investigated, but the results indicated that this test, among those tried, apparently selected the optimal point along the time axis. With all stimuli considered together, it is evident that there is a response in each arm ($p < .01$). When the stimuli were considered separately, the test indicated a significant increase only for the pressure ($p < .02$) and prick ($p < .01$) stimuli in the stimulated arm. In the nonstimulated arm only the response to the prick stimulus exhibited a significant ($p < .01$) increase. In neither arm was there a significant dif-

ference between the increases for the heat stimulus and those for the heat-control stimulus. There is little agreement in the size of the responses in the two arms to the several stimuli.

The Galvanic Skin Response

For each *S* the maximum amplitude of the GSR response within 15 seconds after the onset (2.5 seconds prior to middle of stimulus mark) of each stimulus was measured and the average of the two applications of each stimulus was converted to percentage of the average of the two applications of the prick stimulus. The means of these percentages were 27.7% for heat, 28.1% for heat control, 36.1% for cold, 38.9% for pressure, and 40.0% for tickle. The Friedman test applied to the stimuli just listed was significant at beyond the .05 level of confidence. Including the prick stimulus (100%) increased the confidence level to .01. The Wilcoxon test did not indicate a significant difference between the heat and heat-control stimuli. On the basis of the Friedman test we may conclude the different stimuli have different effects.

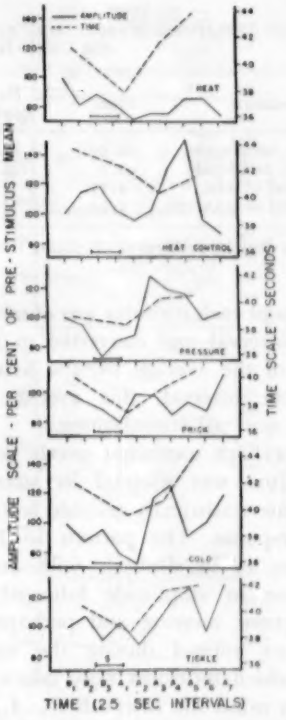

FIG. 18. Mean breathing cycle duration and mean chest-breathing amplitude for each stimulus.

Chest Breathing

The duration of the breathing cycle was measured during each of three successive 5-second intervals following the middle of the stimulus mark and during the 5-second interval beginning 7.5 seconds prior to the middle of the stimulus mark. For each *S* the average of these measures was taken for the two occurrences of each stimulus. The means of these averages are plotted in Fig. 18. It will be seen that the cycle first shortens and then lengthens, in most cases exceeding the prestimulus level by the time of the last measurement interval.

Statistical tests applied to the individual stimuli did not indicate any significant changes. When the data for all stimuli were combined the Wilcoxon test applied to the differences between the 2nd and 4th measurement intervals indicated a significant ($p < .02$) lengthening of the cycle.

The breathing amplitude, plotted along with the cycle durations in Fig. 18, presents an extremely complex picture. The plotted points are the means of percentages obtained in the following way for each *S*. In each of the 2.5-second intervals on the polygraph record the vertical distance from the highest to lowest points of the breathing record was determined. The average of the two appli-

TABLE 15
MEAN AMPLITUDES (PERCENTAGE) AND MEAN PERIOD (ARBITRARY NUMBERS) OF OCCURRENCE FOR CHEST-BREATHING MINIMA AND MAXIMA*

Measure	Heat	Heat control	Cold	Pressure	Prick	Tickle
Min. amplitude	60.0	84.0	61.0	65.5	71.0	69.0
Max. amplitude	92.5	152.0	138.0	151.0	126.0	161.0
Period of min.	2.17	2.00	2.25	1.50	1.83	1.67
Period of max.	5.67	5.67	5.67	5.92	6.17	6.42

* See text for method of obtaining these values.

cations of each stimulus was obtained for each interval and converted to percentages of the average for the first measurement interval, this average being taken over all stimulations.

A perhaps somewhat overly complex procedure was adopted in attempting to locate statistically reliable features of the response. The pattern in Fig. 18 appears to be diphasic with an early decrease in amplitude followed by a subsequent increase and perhaps a return to normal during the intervals over which measures were taken. With this in mind the intervals B_3, A_1, . . . , A_7 were numbered 1, 2, . . . , 8, respectively. For each stimulus the minimum during periods 1, 2, 3, and 4 and the maximum during 5, 6, 7, and 8 were recorded. The number of the interval at which each of these extremes occurred was also recorded, the earliest occurrence being recorded in case of multiple extremes. The means of these values are listed in Table 15.

The amplitudes of the minima do not differ significantly among stimuli by the Friedman test; this test, however, was significant at beyond the .05 confidence level for the maximum amplitudes. The Wilcoxon test was used to compare individual stimuli and indicated that the minimum amplitudes for the heat control were significantly greater ($p < .05$) than those for any other stimulus except cold, for which the response was extremely variable. The significant effect among stimuli for the maximum amplitudes appears to be largely due to the heat stimulus since the Wilcoxon test indicates this response to be significantly less ($p < .01$ in all cases) than the response to heat control, pressure, or tickle.

The only significant difference found when the numbers of the periods at which the minima and maxima occur were investigated was that the maximum for the heat stimulus was earlier ($p < .05$)than that for the tickle stimulus. There was a result, however, which suggests a method for evaluating the response pattern which seems to be in evidence here. The time between the extremes may be represented by subtracting, for each S and stimulus, the period number for the minimum from the number for the maximum. When this was done, the Wilcoxon test indicated that this interval was significantly shorter for cold than for either pressure, prick, or tickle ($p < .05$ in all cases).

In summary, chest-breathing changes appear to consist of an early shortening of the breathing cycle followed by a subsequent lengthening while the amplitude pattern undergoes a diphasic change reaching its minimum about 4 to 6 seconds after stimulus onset and its maximum some 7 to 10 seconds later. These results, of course, do not find

striking statistical support and, further, it must be borne in mind that the variables selected for statistical treatment were chosen subsequent to graphical survey of a set of rather complex response curves.

The Pressure Pulse

The measure taken from the arterial pressure-pulse records was the vertical distance between the maximum up-and-down pen deflections in each 2.5-second interval. After correcting for changes in amplifier sensitivity the corresponding measures for the two applications of each stimulus were averaged for each *S*. Then, for each *S*, the first two intervals (B_1 and B_2) were averaged and all measures were expressed as percentages of this prestimulus mean. These prestimulus levels and percentages were computed separately for each stimulus.

The mean percentages are plotted for each stimulus in Fig. 19. Broadly, the response, except for the heat and tickle stimuli, appears to be a gradual increase in amplitude. The Wilcoxon test applied to the average of of the intervals A_2, A_3, and A_4 indicated that at this point the increase was significant for the heat control ($p < .05$), pressure ($p < .05$) and prick ($p < .02$). The same test applied to the average for the intervals A_6 and A_7 indicated that the early increase shown by the heat-control, pressure, and prick stimuli persisted and apparently became greater through the end of the measurement interval ($p < .02$ for heat control and pressure, and $p < .01$ for prick).

It may be that the effect of the heat stimulus can be interpreted, by comparing the response to the heat-control stimulus, as preventing an increase of pressure-pulse amplitude, i.e., the heat effect is opposite to that of the other stimuli which elicited an increase in amplitude. The Wilcoxon test, however, did not indicate a significant difference between heat and heat control with respect to the test points employed above in establishing the existence of amplitude increases. The argument, then, depends upon the absence of a significant increase in amplitude for the heat stimulus whereas its control does show such an increase.

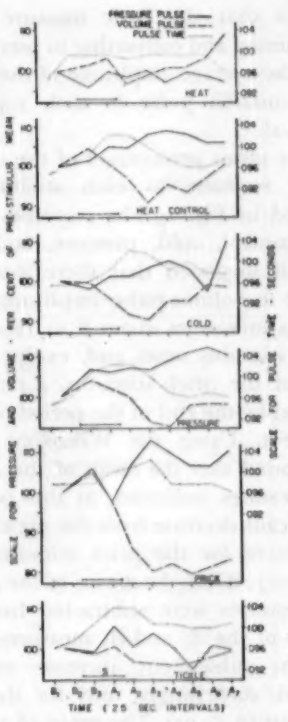

FIG. 19. Mean pressure-pulse and volume-pulse amplitudes and mean interpulse intervals for each stimulus.

The Volume Pulse

The volume-pulse records obtained from the plethysmograph pickups on the right index finger were measured in the same way as the pressure-pulse records

except that the basic measure before combining and converting to percentages was the average amplitude of the largest and smallest pulse in each 2.5-second interval.

The mean percentages of the volume-pulse response to each stimulus are plotted in Fig. 19. The responses to the heat-control, cold, pressure, and prick stimuli suggested that there was a decrease in volume-pulse amplitude reaching its minimum about 8 to 12 seconds after stimulus onset and, except in the case of the prick stimulus, a return to normal by the end of the period of measurement. Using the Wilcoxon test it was found that the mean of the A_3 and A_4 measures indicated, at this point, a significant decrease from the prestimulus amplitude for the prick stimulus only ($p < .02$). When the means of the A_3 and A_4 measures were subtracted from the means of the A_6 and A_7 measures, a significant subsequent increase was indicated, surprisingly, only for the heat stimulus ($p < .02$). The mean of the last two intervals (A_6 and A_7) indicated that the depression of volume-pulse amplitude following the prick stimulus remained significant ($p < .01$) at this point.

The Interpulse Interval

As in previous experiments of this series the pulse rate was measured in terms of the interval between pulses (or "pulse time" as it will be called henceforth) usually in the pressure-pulse records although occasionally this information was more easily obtained from the ballistocardiogram or volume-pulse records. In particular, the average of three successive pulse times was determined for each 2.5-second measurement interval. The measures from the two applications of each stimulus were averaged for each *S* and the means over all *S*s have been plotted in Fig. 19. It will be seen that in general the pulse time increases to a maximum about 4 to 8 seconds after stimulus onset and appears to return to the prestimulus level by the end of the period over which measures were taken. Two tests were applied to the data. The B_1 measures were subtracted from the A_2 measures and the Wilcoxon test indicated a significant increase in the cases of the heat-control ($p < .05$), cold ($p < .01$), pressure ($p < .01$), and tickle ($p < .01$) stimuli. To check the subsequent decrease, or return to normal, the A_5 measures were subtracted from the A_2 measures. Here the Wilcoxon test indicated significant decreases for the heat-control ($p < .05$) and the pressure ($p < .01$) stimuli.

Graphically the response to the heat stimulus appears quite different from that to the heat control and it appears again that the heat effect is to prevent the slowing and strengthening of the pulse which is seen in response to the heat-control stimulus. The argument is still tenuous, however, since comparison of the test points used above did not indicate a significant difference between the responses to the heat and heat-control stimuli.

The Ballistocardiogram

The ballistocardiogram was not easily obtained from all *S*s and was extremely liable to motion artifacts. The records were measured and the measures expressed as percentages of the prestimulus level by the same procedure employed for the pressure-pulse data. It was not possible to obtain readings from all records nor, frequently, to distinguish between pulses and artifacts. As a result many items were missing in the data. The graphs which were obtained suggested that the response might be similar

to the pressure-pulse response; however, the missing data make any interpretation extremely dubious. Various statistical procedures were investigated but no significant changes could be found for the individual stimuli. When all stimuli were combined, the average of the last three 2.5-second intervals indicated a significant increase ($p < .01$) in amplitude of the ballistocardiogram.

Chin and Finger Volume

The polygraph tracings of the volume plethysmographs were measured in terms of vertical deflection from the prestimulus point. These measurements were made at each of the division marks used in establishing the 2.5-second intervals employed for measurement of other responses. The prestimulus point from which deviations were measured was the division line at the beginning of the first of the 2.5-second intervals prior to each stimulus application. These differences were corrected to mm. deflection for a conveniently selected amplifier sensitivity and, thus, the mm. units are actually arbitray units since they refer to the pen deflections which would have been produced by the selected amplifier sensitivity. After this correction had been made, the measures from corresponding points in the responses to the two applications of each stimulus were averaged for each *S*.

The means of all *S*s are plotted in Fig. 20 for each stimulus. It appears that the vasomotor response in the chin and index finger of the contralateral hand is dilation. Since it has not yet been possible to secure satisfactory calibration of the impedance plethysmographs with respect to the absolute volume being measured and since different volumes as well as amounts of vasculature are certainly involved in the finger and chin, we can make no meaningful comparison of the size of the response in these members.

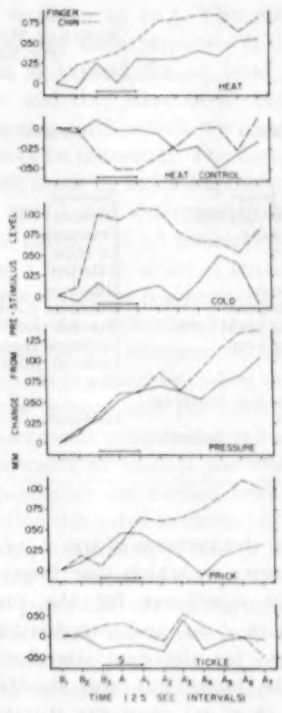

FIG. 20. Mean chin and finger volume changes from prestimulus level for each stimulus. The mm. units refer to recording pen deflections adjusted to an arbitrary amplifier sensitivity.

The dilations were checked at two points by means of the Wilcoxon test. For the first of these the A_2 and A_3 measures were averaged and a significant finger dilation was indicated only for the pressure stimulus ($p < .01$); in the chin the dilation was significant for the pressure ($p < .05$) and heat stimuli ($p < .02$). The differences between the heat and heat control were not significant in either case. The second test was

TABLE 16
RANKS OF MAGNITUDE OF EFFECT OF STIMULI FOR EACH RESPONSE VARIABLE

Response	General Effect	Stimulus					
		Heat	Heat control	Cold	Pressure	Prick	Tickle
Pressure pulse	Increase	1	4	3	6	5	2
Ballistocardiogram	Increase	2	4	1	6	5	3
Volume pulse	Decrease	1	5	3	4	6	2
Pulse time	Increase	2	4.5	3	6	1	4.5
Finger volume	Dilation	2	6	4	1	3	5
Chin volume	Dilation	2	6	1	3	4	5
Skin resistance	Decrease (GSR)	1	2	3	4	6	5
Breathing cycle time	Shorten then *lengthen*	1	6	2	4.5	4.5	3
Breathing amp.	Decrease then *increase*	1	5	3	4	2	6
	Interval from min. to max.	2	3	1	5	4	6
Muscle action potential, stim. arm	Increase	3	1	5	6	4	2
Muscle action potential, non-stim. arm	Increase	2	1	4	3	6	5

made for the averages of the A_5, A_6, and A_7 measures in which case finger dilation was significant for the pressure stimulus ($p < .01$) and chin dilation was significant for the heat stimulus ($p < .02$). The latter was also significantly greater ($p < .01$) than the dilation (if any) for the heat control.

These results indicate that appropriate tactual stimulation produces a vascular dilation, an effect which is opposite to that of the auditory stimuli used in the previous experiments. Recalling that the volume-pulse response was in general a decrease in amplitude while the pressure pulse was increasing and the pulse duration lengthening, it may be that here the elastic limit of the vessel walls is being approached during the dilation. In this case the pulsatile volume changes in the periphery might be expected to be of small amplitude.

The Pattern of Responses

The features of each of the response variables with respect to the individual stimuli have been reported in the above section. This section is concerned with grouping the response variables in terms of their patterns with respect to the set of stimuli. When dealing with the individual responses, certain points in the record were selected and statistical tests were made to determine whether there was a significant effect at these points due to the various stimuli. In order to group the response variables they were considered as judges whose task was to assign ranks to the stimuli in the order determined by magnitude of effect. In Table 16 the ranks determined in this way are listed. In selecting for ranking one of the several measures used for statistical evaluation of a response variable, the rule followed was to rank the means of that measure for which the largest number of significant results had previously been found.

The smallest rank was assigned to the stimulus to which the smallest effect was seen in each response. In general it will be clear what is meant by largest and smallest effect but in the case of three responses the rationale for the order of

the ranking must be clarified. The finger and chin volume changes were seen to be, in general, dilations. This is interpreted as a decrease in vasomotor tone, so that the *largest* dilations receive the smallest ranks. Breathing-cycle duration ranks are also smallest for the largest changes. There is no clear rationale for this treatment of breathing time except that it was found to yield definitely better agreement with the ranks for most other responses.

In order to evaluate the agreement of the responses as judges, the Friedman concordance-of-ranks technique was employed with various groupings of the responses. The selection of these groups was at least partially made from inspection of the data and hence the statistical risks may actually be greater than indicated in what follows. There is no way, however, to correct the confidence levels so as to guard against this possibility. In consequence, the results of the statistical tests are reported without modification and this shortcoming must be kept in mind in what follows.

As there were six stimuli, or treatments, the chi-square estimate, χ_r^2, obtained by the Friedman test has in all cases five degrees of freedom. Since it will be convenient to report only the chi-square estimates for the concordance-of-ranks test with the various sets of responses we note here that a chi square of 11.070 is significant at the .05 confidence level, 13.388 is significant at the .02 confidence level, and 15.086 at the .01 level.

The most natural grouping of responses is the cardiovascular group: pressure pulse, pulse time, ballistocardiogram, volume pulse, finger volume, and chin volume. The χ_r^2 for the first four of these was 12.2 and when the finger and chin volume ranks are included this value increases to 12.5. The rank concordance for these responses is thus significant at beyond the .05 level of confidence and they have been taken as a basic group with which the other variables may be compared. The comparison has been made by including the variables to be tested and determining how the statistic changes. If χ_r^2 increases upon including a response or set of responses we conclude that there is agreement between these and the cardiovascular group and conversely if χ_r^2 decreases. In only one case was there a decrease: when the cardiovascular group and the muscle-action potential ranks were considered together χ_r^2 decreased to 10.1. If the muscle action potentials are ranked from largest to smallest this value becomes 12.0 which, although significant, is still a decrease.

Where all response except the muscle action potentials are considered, χ_r^2 is 22.2. Including the muscle action potentials decreases this value to 19.5 or to 19.6 if ranks are assigned in the opposite direction.

It appears that, in terms of magnitude of effect, the somatic responses, with the exception of the skeletal-muscle tensions, behave in similar ways with respect to the stimuli employed in this experiment. The most conspicuous consistency, looking at Table 16, is among the ranks for heat and cold. In these two stimuli, particularly heat, are found the preponderance of low ranks, i.e., small effects. It would appear that the thermal effect here is to prevent or, put another way, is opposite to, the "normal" effect, if we may consider the latter to be represented by the response to the heat-control stimulus.

Considering only the autonomically controlled components, we may say that the response varies quantitatively from stimulus to stimulus, though, among these, there may be pattern changes we

TABLE 17

VARIABILITY OF RESPONSE MEASURES IN RELATION TO MAXIMUM MEAN RESPONSE

Response Variable	Repetition Experiment			Intensity Experiment			Cutaneous Stimuli Experiment		
	Max. Mean Response	*SD*	$\frac{SD \times 100}{\text{Max. Mean}}$	Max. Mean Response	*SD*	$\frac{SD \times 100}{\text{Max. Mean}}$	Max. Mean Response	*SD*	$\frac{SD \times 100}{\text{Max. Mean}}$
MAP Right arm									
a-Response				94.6	59.8	63			
b-Response				44.6	34.2	77			
MAP Left arm									
a—Response				56.1	29.1	52			
b—Response				31.6	26	62			
MAP % prestim.									
Stimulated arm							204	322	157
Other arm							62.0	85.6	137
Skin Resistance (GSR)	20.5	9.2	41	37.4	19.7	53			
$GSR+GSR_{Max}$							1.00	.33	33
GSR latency (Sec.)	3.20	1.87	58						
GSR reciprocal latency (1/Sec.)	.459	.225	48	.361	.308	85			
Interpulse interval (ms)	14.8	60.8	410	75.4	87.0	116	77.1	73	95
Volume pulse	2.50	3.50	140						
Volume pulse									
% Decrease	27.5	26.0	95	31.0	30.9	100	18.9	15.1	82
Pressure pulse									
% Increase	20.0	28.7	143	56.0	61.9	110	7.2	10.1	106
Finger volume	2.21	1.99	90	2.60	2.41	92	.525	.817	155
Finger volume									
Latency (Sec.)	8.42	5.75	68						
Finger volume									
Latency reciprocal (1/sec.)	.171	.135	79						
Chin volume				2.62	3.02	115	1.72	1.20	70
Systolic pressure	3.1	5.7	185						
Breathing cycle									
Time (Sec.)	.33	.62	184				.61	1.13	185
Breathing rate (cycles/min.)				1.32	3.05	231			
Breathing amplitude	2.36	4.50	190	1.34	2.45	182	1.32	1.28	97

have not detected. Where the skeletal-muscle responses are included there is evidence of a change of pattern according to stimulus, for there seems to be no correlation between skeletal-muscle and autonomic responses under these circumstances.

The pattern of the autonomic responses is similar to that produced by the auditory stimuli except for the finger volume effect. The tactual stimuli produce an increase rather than a decrease of finger volume.

VI. THE VARIABILITY OF RESPONSE VARIABLES

Little has been said of variability in the foregoing sections. Yet it necessarily has been considered in all the significance tests that we have applied. Although they involve no computation of a dispersion parameter, the nonparametric tests rest upon the comparison of experimentally produced effects with uncontrolled variation. (In certain of these tests, even the effect of N is hidden, though powerful.)

While we do not propose to duplicate our analysis of central tendencies with a similar study of variability effects, we will assemble here (Table 17) some information on the dispersions of the several variables in our three experiments and venture certain interpretations of them. A standard deviation, when compared to experimental effects, can be interpreted as a measure of the experimenter's failure: it represents the enemy territory he has not yet been able to subdue.

Many kinds of standard deviations could be computed from our data: *SD*'s corresponding to the total variance of readings, to the total variance of responses as we have defined them, to the variance of individuals in either respect, to the variance of individuals within conditions, to the variance from all sources within conditions, or the residual variance, to give but a partial list. We have chosen to present in Table 17 a set of *SD*'s which represent the total variance of the data which the experimenter had to analyze. These *SD*'s apply to the variance of responses: differences, that is, between certain poststimulus readings and certain prestimulus readings, as defined in the three preceding sections of this report. (Therefore they do not represent the term which would be treated as "error" in analysis of variance.)

The units in which the various responses are expressed are necessarily various. Some, as indicated in the table and explained in the text, are in time and rate units, some are in terms of percentage change, some (the MAP of the second experiment) in log microvolts; systolic pressure is in arbitrary pressure units and the others are in arbitrary deflection units, comparable only with *Ss* and between conditions. Consequently, direct comparisons are limited. To permit comparison of variability among all the response variables we have adopted the scheme used for comparing condition means; i.e., we have expressed the standard deviation as a percentage of the maximum (condition) mean response in each experiment.

In Table 17 there are certain implications about methods of quantification, since we have tried out several for some of the variables.

For muscle action potentials the percentage of prestimulus-level treatment may be compared with the increase-in-log-microvolts technique. The former gives about double the *SD* of the latter (in terms of percentage of maximum mean), in spite of the fact that the maximum

mean response is widely different for the stimulated arm and about equal in the nonstimulated arm in the two experiments. This fact points to an inflation of standard deviation by the technique of expressing MAP reactions as percentages of prestimulus level, an inflation it would be well to avoid.

For the GSR, the lowest *SD* in percentage terms is in the third experiment. The technique used there (expressing each of *S*'s responses as a percentage of his response to a specified stimulus) may serve better to exclude individual differences in reactivity than does the more conventional procedure of using *S*'s prestimulus resistance as a percentage base. The scheme might also serve well for other response variables.

Putting volume-pulse responses into terms of prestimulus level reduces their standard deviation (in terms of our percentage figures). The reduction no doubt comes from the elimination of certain structural parameters of the individual. (This is the reasoning behind the practice.) From a statistical point of view, however, the use of percentages of this sort is not ideal, for this or any other response variables. There is danger of producing a very skewed distribution, possibly suggesting false conclusions. But until better methods are devised, one may use this device and use only such inferential methods as do not depend upon distribution form.

Examining Table 17 as a whole, one finds a tendency for each response variable to maintain about the same relative position, with respect to the other, in amount of variability (in our percentage terms). An exception which stands out is the interpulse interval in the first experiment, which has an extremely large percentage. Since the absolute measures may be compared here, it is evident that it is the maximum mean response which is out of line here as compared with the other two. Inferring from Fig. 12, one would expect it to be about twice as large as it is. The explanation may be that in the latter experiments finer units were used in the original measurements of the records of this variable.

Generally it seems that response variables which are periodic in nature (breathing and pulse variables) have a higher variability than do the aperiodic variables (GSR, MAP, and the volume measures). We may speculate that pulse and breathing systems are always maintained in a fairly high state of activity and are therefore subject to many inputs which the experimenter does not control. Where a low level of functioning is normal, the experimenter may control a greater proportion of the determining factors. These suggestions can be only tentative, because improved techniques may change the relationship.

Because they are easier to quantify, time measures might be expected to have lesser variability. On the whole, however, they seem to have no particular advantage over comparable amplitude measures.

In general, the expectation is borne out that our experimental results are most often significant where the *SD* (percentage) is low. In designing future experiments, it may be profitable to take account of such differences in variability as show up here.

VII. SOMATIC RESPONSES AS THE OUTPUT OF RESPONSE SYSTEMS

The results clearly show a lawfulness in somatic responses. The stimuli used regularly produce changes which show a certain variability but can by no means be called random.

First we should like to point out the obvious: that stimuli produce somatic responses even when there is no manifest movement. The body, it may be said, reflects external conditions with some sensitivity. Some of our stimuli were not unusually intense, yet even these elicited

changes in the organism. As a person probably encounters similar brief stimuli many times a day, we may suppose that he is in a continuous and fluctuating state of somatic upheaval so long as he is awake.

These surges of action, quickly aroused and slow to expire, permeate the organism so far as our observations are carried. For all the stimuli and situations we have tried there are signs of change in each of the response variables we recorded. Circulation, respiration, sweating, and striped muscle are all affected by each kind and strength of stimulus. (For some of the cutaneous stimuli, however, we cannot assert this with a presentable confidence level.)

Viewing a response which seems to spread as far as the nervous system penetrates, one may be tempted into speculation on its biological import. Can it somehow be fitted into the biological philosophy of equilibration by homeostasis? With a certain glibness of diction the result is not hard to achieve. One may allege that our stimuli are environmental stresses which the organism resists by countervalent interior forces. Or that our responses serve to overcome some unseen internal threat which the stimuli have released. These conceits are indeed picturesque, but offer more of a *mystique* than a rationale for the events. Until all the terms have independent meaning, they cannot be good metaphors; their substance is that the organism assumes a new state following certain events, which is the given fact.

In the unadorned account it is hard to see anything homeostatic about these somatic responses. They do, of course, eventually disappear after the stimulus has ended, and they may contain in themselves seeds of their own destruction, in that they activate homeostatic mechanisms. But this is not a necessary supposition, unless, of course, we wish to count any restoring force as homeostatic, and speak, for example, of the homeostasis of a displaced pendulum.

There still remains the question of biological significance, but we are scarcely prepared to offer an embracing hypothesis about it. Whether the question is the phylogeny of responses or their ontogeny, or whether it concerns an effect on the fortunes of the organism, it seems to us the present need is for narrower hypotheses which will point to many new observations. Given hypotheses about the external conditions and biographical events which control the responses, and about their effect upon the particular kinds of external response, one would be able to carry out experimental studies for their verification.

Such hypotheses would need to be particular in another way too. For it seems to us that our results show there is a near limit to what may be said about the somatic responses in general. Though such responses spread through the organism they are nevertheless not alike. Studies by Lacey *et al.* (32), Malmo *et al.* (35), Wenger (51) and Jost and Sontag (28) have found that autonomic responses tend to be peculiar to the individual. The many clinical observations of the psychosomatic school are also similar in their import. In the present series of experiments the question of stimulus control of response differences was at issue, and we are able to conclude that varying the stimulus in certain ways will change not only the size of the response but also its character. Our brief noise stimuli produce what we call the *N*-response, consisting of a circulatory response dominated by vasoconstriction and cardiac deceleration, slow deep breathing, galvanic skin response, and

brief skeletal-muscle tension. This complex becomes weakened by repetition and its emphasis shifted to the respiratory component. Our cutaneous stimuli and the deep pressure stimulus of arm compression produce, however, a circulatory response dominated by vasodilation, and there is evidence that the cutaneous stimuli among themselves produce different effects. Further it seems improbable that our few stimuli and treatments have elicited all the response variations that might be brought to light.

We have no grounds, of course, for dubbing any of our responses "fear," "startle," "anxiety," "anger," or anything else in the ample psychological vocabulary, unless we were to define the term by this operation. Rather than add to present confusion about these words, we preferred to use a neologism—the *N*-response—which has the clear virtue of previous emptiness. The domains of the other responses described do not seem clear enough yet to deserve names. Names which the *S*s might call their responses we did not think it worthwhile to worm out, since we felt sure the *S*s' verbal responses would be quite indefinite.

It is hardly possible to match our varieties of response with the sympathetic-parasympathetic anatomical divisions. The *N*-response has many features usually considered to be sympathetic in origin, but it also has some characteristics usually taken as parasympathetic signs. In the cutaneous response there are also mixed symptoms. It seems that all our responses result from different combinations of the two divisions, with the dominance differing from site to site and from one stimulus to another.

So far as response patterns are different the state of a single response variable cannot serve as an index of the size of a response or the general condition of the organism. On the other hand, where one has reason to believe that the responses being compared are reasonably similar, as they are under variations of auditory intensity, one response variable might serve well. To make decisions of this sort in general clearly requires more knowledge than so far exists.

Even with different types of response one might hope to reduce responses to a common denominator, to set up, as Duffy (13) has proposed, a quantitative as well as a qualitative specification. There is the possibility of totaling the energy consumption of all responses within a given period, though there seems to be no way of measuring this for events of short duration. Or, to be still more speculative, one might construct a multidimensional manifold for the response variables and by vector addition discover the distance of a response from a certain point of origin. Whether there is anything to be gained by such conceptions remains to be found out. It is possible that a complex variable so defined might have certain relations to the behavior of the individuals, relations in which the individual components of the response are interchangeable. More promising, it seems to us, is the possibility of behavioral relations to specific patterns of response. As a matter of procedure it would seem better to make the hypothesis that a given somatic condition increases the probability of one external response and decreases it for another, for the hypotheses about general effects might lead to experiments which overlook specific ones.

When we are able to say, as we are, that a certain set of somatic changes will follow upon a given stimulus, we have identified a physical response system in the organism. Considering the system for our *N*-reaction, it is evident that a sys-

tem in this sense does not correspond with any organ system of the body nor yet with the gross structural divisions of the nervous system. The receptors, the neural structures, and the effectors involved are each activated by a prior element in the chain, and, except for the ultimate one, activate the next succeeding element. Local characteristics of the elements will determine the character of the action, transducing it from one sort of force to another. Each element may be said to transmit the disturbance to the next, but the transmission is also a distribution. For the disturbance which enters the body at one point, say the auditory receptor, reaches many points on returning to the periphery. One is tempted to compare the spreading to the propagation of waves in a homogeneous medium, but the picture is a poor one because the distribution changes with known conditions. A better picture would be that of a branching network, which the nervous system actually is, with the possibility of transmission at any point being able to vary somewhat independently of that at any other.

The temporal course of events at the ends of the various branches suggest something of their dynamic characteristics. To gross inspection, at least, many of the response curves (with respect to time) look much alike though their time axes are quite different. Those responses which are single phased have rising periods that are comparable to their latencies and decay periods (which are very much longer). They resemble the responses of second-order systems with critical or more than critical damping (47). The difference in time scales bespeaks a much longer natural period for some branches of the system than for others, suggesting that these have a much greater inertial factor. The longer latencies for these branches could also result from a large inertial factor in the elements preceding the terminal ones if there are thresholds which must be reached along the way. A disturbance transmitted over a row of slow-moving pendulums by one setting the other into motion would have a rather long transmission time. The time differences in the several branches of the system lead to the conclusion that parts of the organism keep fairly well in step with external events, while other parts lag far behind and still others are intermediate. In the slower parts, further, the effects of external events widely separated in time would be superimposed, and their condition at any one time would reflect all that has happened to the organism over a considerable stretch of time. One might say that temporal discrimination in those parts would be very poor.

When different stimuli, auditory and cutaneous, produce different responses, we evidently are dealing with response systems which are entirely separate in their early stages (in the receptors and afferent pathways), but overlap in their subsequent ramifications. That the overlap is not complete should occasion no surprise, for only in a case of very special engineering would the same effect arise from signals introduced at different places in a compound network. We do not have that simple case, nor indeed the other one in which two or more systems are isolated from each other throughout. In other words, we do not have simply one or more "centers," each dictating to its own family of effectors. Something of the original stimulus differential is preserved up to the end of the chain. In our experiment which compared the effects of auditory intensities, on the other hand, there was no initial place differentiation of the stimuli (or

very little of it). These stimuli turned out to activate the same response system, in different degrees. An input intensity difference remained as an output intensity difference to a first approximation. Whether stimuli to the same receptor not having the gross separation provided by the different "analyzers" but differing, say, in temporal pattern, would activate somewhat different response systems, is a question for further experiment. A study of lie-detection methods carried out in this laboratory shows a good deal of resemblance between responses to interrogation and the responses to simple auditory stimuli of the present experiment, but the untried stimulus variations are, of course, quite numerous.

The case of changing response with repetitions of a stimulus is of different theoretical aspect. Here the stimulus is objectively the same but has different effects. Why should it on later occasions activate a different response system, or, if one prefers, the same system in a different manner? There are two general possibilities: First, the system might have a residual of activity from the previous stimulus, and the system would approach, no doubt asymptotically, its limit of response. Further, because residuals would evidently be different in the various branches of the system by reason of their different time constants, the pattern of response would change. Such an effect ought to exist, but there are several reasons for doubting that it altogether explains the present results. Adaptation effects in certain branches, for one thing, have been demonstrated over periods of days. In the present data, moreover, the slowest response elements show the least adaptation, while on the above hypothesis the reverse should be true.

We must consider, therefore, a second possibility: that each response leaves behind it a reversible change in the parameters of the response system. If these changes are also different in the several branches of the system, we should get the changed pattern that we do. This is a very distressing sort of hypothesis, for unless "changed parameters" can be identified in some way, it is no more than verbal legerdemain. The problem, of course, is a central one which physiological psychologists usually avoid facing as long as possible: the nature of semipermanent changes following response: the learning problem.

We can make but a slight contribution to the meaning of "changed parameters." Our data show that the changes are not all in one place. Since the response elements behave differently they must be subject to different parametric changes in the several branches. In the case of one response element, the GSR, we do have a measure of the condition of the tissues involved prior to the application of each stimulus, and the response in general is closely bound to this tissue condition. The facts led us to offer a hypothesis about the role of electrotonus or polarization of the tissues. Studies of this sort of relation, possibly extended to other response elements, might yield something more than a circular explanation. Of what, if anything, these somatic responses have to do with the determination of the individual's behavior, we can, of course, say nothing on the basis of these experiments, though we share the common expectation that they will turn out to be of some importance. Definitive investigations of the topic require basic knowledge of the controlling factors such as we have studied here.

VIII. SUMMARY

This monograph reports a series of experiments on the somatic responses to external stimuli when there is no external movement required of the *S*. Recordings were made of numerous features of cardiovascular, respiratory, dermal, and skeletal-muscle responses. Topics investigated were:

1. The interrelation of various cardiovascular response measures.

2. The stimulus effect of blood-pressure measuring techniques.

3. The effect of stimulus repetition upon the various response elements and their interrelation.

4. The responses as functions of auditory stimulus intensity.

5. The nature of responses to a variety of cutaneous stimuli.

The direction and temporal course of the response was ascertained for each response element in each situation and the response magnitude was related to the stimulus condition.

The principal conclusions are:

1. There is a common type of circulatory response in which many aspects of the cardiovascular system are affected by peripheral vasoconstriction.

2. Strong pressure on the arm such as used in blood-pressure measurement produces a general vasodilation and other circulatory changes.

3. Repetition of a stimulus diminishes most elements of the response, though at different rates, but increases the respiratory response to a 90-db. stimulus. The pattern of response is changed, therefore, by stimulus repetition.

4. As the intensity of an auditory stimulus increases from 70 to 120 db. the somatic response increases more than proportionally, the log of the various response measures being somewhere near proportional to the decibel intensity. The response pattern does not change greatly with increasing intensity. There are some interaction effects between repetition and intensity, most conspicuous in the fact that repetition of the 120-db. stimulus decreases the respiratory response, while repetition of the lower intensities increases it.

5. Cutaneous stimuli produce a somewhat different response pattern from auditory stimuli and seem to determine different responses among themselves according to their objective character.

In general there is evidence of a number of different but overlapping response systems whose differential action is related to the nature of the stimulus and whose characteristics can be modified by stimulus repetition.

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